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CARDIOLOGY

BY

WILLIAM EVANS
M D, D Sc, F R C P.
PHYSICIAN TO THE CARDIAC DEPARTMENT OF THE LONDON HOSPITAL, PHYSICIAN TO OUT-PATIENTS OF THE NATIONAL HEART HOSPITAL, CONSULTING CARDIOLOGIST TO THE ROYAL NAVY

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TO

SIR JOHN PARKINSON

In grateful recognition of his teaching, encouragement and friendship

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PREFACE

STANDARD text-books on diseases of the heart are already available so that it would seem unnecessary to add another one. This book is intended to fulfil a purpose other than that of providing an exhaustive account of all the disorders peculiar to the heart and blood-vessels; it is meant to serve the need of the medical student who seeks a more concise treatise on this subject in preparation for a qualifying or higher examination. Indeed the initiative for this work was provided by post-graduate students who asked me to place on record a series of lectures given to them at the London Hospital. This response to their request is made in the hope that it will prove helpful to them and to those who succeed them.

To the busy medical practitioner, whose opportunities to consult larger works on cardiology may be limited, this book may serve as a convenient substitute.

The reading of other standard text-books on this subject has naturally influenced me in my writing, and I freely acknowledge the help I have gained from these.

Mr. William Dicks, Senior Technician to the Cardiac Department of the London Hospital, has collaborated in producing most of the records illustrated in this book, and Mr. John King has helped in the printing of negatives.

WILLIAM EVANS

September, 1948.

CHAPTER 1

HISTORY TAKING AND PHYSICAL EXAMINATION

HISTORY TAKING

THE INTERROGATION of a patient suspected of cardiovascular disease does not differ materially from that adopted for any other illness, the general plan is the same but obviously the nature of the questions must vary. In the case of no other disease is it more desirable for the patient to be allowed to recount his own symptoms spontaneously and not in response to leading questions. Adherence to this custom is especially important when information is sought concerning pain in the chest. Of equal importance, too, is the allotting of sufficient time for a detailed record of a patient's symptoms. Hardly any symptom can be regarded as irrelevant in the diagnosis of a cardiovascular disorder, in estimating the prognosis, and in determining the patient's future share in physical activity.

DURATION OF THE ILLNESS

There are many reasons for knowing the time of onset of the illness. It is necessary to ascertain how rapidly the condition is progressing. Neglect to conduct an earnest inquiry about past symptoms often results in a failure to discover a symptom-free period during a stage of the illness, leading to a false estimate of the time of its onset Again a knowledge of the duration of symptoms facilitates judgement on the benefit gained from past treatment, and on the future outlook.

HABITS

Apart from knowing a patient's occupation an itinerary of daily activities should be available, providing an index of cardiac efficiency and a means of evaluating the capacity for work in the future. Thus, ability in the past to undertake a strenuous occupation helps to assess the degree of cardiac injury and the response to a period of rest. An acquaintance with a patient's home responsibilities and financial obligations is necessary, for they provide a measure of his opportunity for resting during a period of treatment and during actual employment. Information regarding customary recreation should also be available when advice on future activities is outlined The influence of injurious agents which contrive to aggravate the symptoms of cardiovascular disorders must come under review, so that excesses or dietary indiscretion may be corrected.

PAST ILLNESSES

In a search for the cause of cardiovascular disease every record of past ill-health must receive attention. When valvular disease is present inquiry might be made concerning the appearance in the past of any of the manifestations of rheumatic fever, *namely, acute arthritis or chorea, but there is need to emphasize that the* presence or absence of such history must on no account sway an opinion based on the findings of clinical examination. In the case of heart pain or aortic incompetence a previous history of syphilis is significant. When a congenital cardiovascular

disorder is suspected, evidence that a heart murmur was present in infancy or early childhood is of value.

RECORD OF SYMPTOMS

A patient with cardiovascular disease may only tell of his chief symptom which often masks the lesser ; thus if dyspnoea is severe it will overshadow most other symptoms including pain After a record has been made of the presenting symptom, therefore, it is necessary to elicit by direct interrogation, information about other symptoms relative to the suspected condition or the outcome of heart failure. Pain in the chest and the effects of heart failure are the symptoms most common to a patient with cardiovascular disease, but others associated with hypertension, renal disease, and certain vascular abnormalities are not uncommon. With regard to dyspnoea it is necessary to know whether it occurs at rest, or is only induced by effort, and whether it is exhibited as attacks at night (paroxysmal nocturnal dyspnoea or cardiac asthma) in this connexion information about the number of pillows customarily used by the patient is of special value Palpitation, flatulence, anorexia, indigestion, syncope, cough and haemoptysis are other and ancillary symptoms.

PHYSICAL EXAMINATION

The cardiovascular system is not the only one to claim attention when disease of it is either present or suspected, but the nervous, pulmonary, digestive, renal and endocrine systems should also be examined. Neglect of these has often led to failure to discover some associated or complicating lesion, and this in turn has resulted in wrong diagnosis, prognosis, and treatment. A scheme for the clinical examination of the heart may be discussed under four headings.

GENERAL EXAMINATION

The patient's physical development and nutrition should be noted. Plethora gives concern in the treatment of cardiovascular disease, and leanness may be important in diagnosis. The appearance of the face may disclose cyanosis, pallor, or anaemia Clubbing of the fingers and toes, developmental anomalies, either physical or mental, have greatest significance when congenital heart disease is suspected. Examination of the thyroid gland must never be omitted for its enlargement will give rise to a natural search for the signs of thyroid toxaemia. To notice the breathing and the posture adopted to provide for the greatest respiratory ease, is of first importance The height of the head-rest, estimated as the number of pillows necessary to promote sleep, supplies an index of the severity of cardiac dyspnoea. Other objective symptoms of heart failure should be sought, namely excessive distension of the veins in the neck, enlargement and tenderness of the liver, ascites, oedema of the lower extremities, and crepitations or evidence of fluid at the lung bases. Examination of the urine, and of the optic discs and retina, must never be neglected in any patient with hypertensive heart disease. Changes in the central nervous system may sometimes explain certain cardiovascular signs, and especially those caused by syphilis.

EXAMINATION OF THE PULSE

Whenever disease of the heart is suspected a preliminary examination of the pulse will frequently yield valuable information. Thus, when a collapsing pulse has been

discovered, a special search for aortic incompetence will naturally follow. Again if the pulse is irregular from auricular fibrillation the presence of mitral stenosis, hypertension, or thyroid toxaemia will be kept in mind. During examination of the radial pulse it is first necessary to determine the rate and then the rhythm. If the rhythm is irregular the nature of the irregularity should be defined in terms of sinus arrhythmia, extrasystoles, paroxysmal tachycardia, auricular fibrillation, or heart block. The character of the pulse should be noted and an opinion expressed as to whether it is small or collapsing. Sometimes it may be possible to make out that alternate beats are small (pulsus alternans). A record of the blood pressure is necessary in every patient suspected of cardiovascular disease The following are recommendations on the methods of measuring arterial blood pressure.

The instrument—The equipment used for measuring arterial blood pressure, whether of the mercurial or aneroid type, must be in good condition and in the case of the aneroid type, should be frequently calibrated against a standard mercurial manometer The mercurial type is the most dependable

Position of the patient.—The patient should be allowed time to recover from any recent exercise or excitement, and must be comfortable either in the sitting or reclining posture There should be no constriction of the arm or interference with the armlet by any clothing

Application of the cuff—The cuff must be standard size and with its rubber bag at least 12 cm. wide The cuff, completely deflated, should be applied with the middle of the rubber bag over the inner side of the arm, and its lower edge 1 inch above the bend of the elbow It should fit closely and evenly around the arm to ensure against bulging at the sides when inflated, particularly in obese subjects

Determination of the systolic pressure—With the stethoscope applied lightly and evenly over the brachial artery just below the cuff, the bag is inflated quickly to a pressure about 30 mm above the point at which the sound of the pulse disappears, and deflated slowly The systolic pressure is the level at which successive sounds are first heard

Determination of the diastolic pressure—With the pressure in the cuff continuing to fall slowly and uniformly, the sound increases to its maximal intensity and then decreases, at first gradually and later suddenly The point where the loud clear sounds change abruptly to the dull and muffled sounds should be taken as the diastolic pressure.

Special conditions.—In auricular fibrillation only approximate blood pressure readings can be obtained, the systolic value should be taken at the point at which the majority of the beats appear, and the diastolic (if possible to obtain) at the point where they become muffled Alternation in the strength of the beats (pulsus alternans) should be carefully looked for, this must be distinguished from the alternating values given by alternating extrasystoles (pulsus bigeminus) In aortic incompetence the diastolic pressure is marked by a less obvious change in the quality of the sounds If the pulse feels different on the two sides, the blood pressure should be taken in both arms When a raised blood pressure in the arm is unexplained it should also be taken in the leg when coarctation of the aorta may be discovered For recording blood pressure in the leg the cuff and its covering should be wider and longer, the patient should be in the prone position with the leg extended and the sounds auscultated over the popliteal artery below the lower edge of the carefully applied cuff.

The extent of arterial pulsation in the neck often provides a clue in diagnosis. Pulsation in other arteries may also need to be tested, and especially in arteries to the lower extremities when ischaemia of one of the limbs is suspected. The state of the arterial wall can be ascertained by palpation, but hardening or tortuosity of the radial and brachial arteries must not weigh in prognosis because it is never an index of the condition of the more vital arteries such as cerebral or coronary.

EXAMINATION OF THE HEART

The methods applied to the clinical examination of the heart are conveniently considered under the headings of inspection, palpation, and auscultation. *Percussion*

Auscultation

Although convention has decreed that auscultation shall be carried out in the four areas, mitral, pulmonary, aortic and tricuspid, whenever a murmur is present, especially if it is atypical, it is necessary to listen in a wider circuit, particularly around the mitral area and along the lateral borders of the sternum. Auscultation over the xiphisternum should become routine procedure because of the frequency with which triple rhythm exhibits itself to best advantage in this area. A systolic murmur is often audible in the right supraclavicular area, but it is only of significance when it is long and appears in the presence of an aortic murmur. Primary attention to the character of certain loud murmurs often leads to neglect in timing the murmur and failure to discover more obscure ones. In order to ensure proper attention to each part of the cardiac cycle, a scheme is outlined which compels a study of each separate phase of the cycle characterized by the heart sounds. Thus, when the stethoscope is applied to each auscultatory area the observer should provide an answer to the following questions

1. **Do I hear the first sound ; is it accentuated or does it show splitting ?**
2. **Do I hear the second sound ; is it accentuated or does it show splitting ?**
3. **Do I hear more than two sounds ; is the extra sound in front of the first sound or is it near the second sound ?**
4. **Are there any murmurs connected with the first sound ?**
5. **Are there any murmurs connected with the second sound ?**
6. **Is there a murmur following the third heart sound ?**

Simple as this self-catechism may seem it is not possible to exaggerate its importance in the diagnosis of heart disease in so far as auscultation applies.

The presence of pericardial friction sound should be kept in mind Of some importance is the influence of respiration on the intensity of murmurs, and auscultation needs to be carried out when the breath is held, during quiet respiration, and at the end of deep inspiration. Any manoeuvre by the patient or the observer which changes or accentuates the murmur has to be practised. It is a first rule that auscultation should be carried out with the patient in the erect and recumbent postures. Whenever a mitral presystolic murmur is suspected auscultation should be carried out immediately after exercise and with the patient reclining on the left side. When an aortic diastolic murmur is sought the patient should sit upright while auscultation is carried out by direct application of the naked ear to the chest at the lower end of the sternum with the breathing halted at the end of expiration

ACCESSORY METHODS OF EXAMINATION

Sphygmography

The examination of the pulse-wave from a graph traced by a Marey or Dudgeon sphygmograph is no longer carried out in the routine investigation of a patient, but the application of these findings in the past has explained certain changes in the pulse which are determined by palpation.

Polygraphy

It was with the aid of the polygraph, combining as it does a radial pulse tracing (sphygmogram) and a record of the pulsation of the jugular vein (phlebogram),

as a means of detecting the size of the heart may be discarded as obsolete; it is inaccurate even for the delineation of a grossly enlarged heart. The estimate of cardiac enlargement gained from the finding of a displaced apex beat is always more precise than the evidence collected from percussion of the cardiac area. Adherence to this traditional method of examination can never advance our knowledge of cardiology, and since it inevitably deceives both teacher and student of clinical methods, it may produce harmful effects. Even in the discovery of an aortic aneurysm, percussion can seldom be the only physical sign, for when the vascular swelling has become so contiguous with the chest wall as to give rise to dulness on percussion, its pulsation will be both seen and felt. It is not to be denied that when the pericardial sac has become distended with fluid, percussion will implement the clinical diagnosis, but even in this instance the lesser degree of pericardial filling at the beginning of the illness can only be discovered on x-ray examination.

Inspection

The localization of the apex beat by inspection is only approximate, and sometimes it is not possible. Inseparable from the search for the apex beat is the search for scoliosis which is best told by inspection of the front of the chest. Thus, in the common type, displacing the apical impulse outwards, the left shoulder is higher than the right, and the right nipple lower than the left, and the right chest is also more prominent than the left. Bulging of the left side of the chest may show in children as a result of gross cardiac enlargement. Peri-apical systolic retraction, although without much significance, may sometimes be present, and similarly retraction of intercostal spaces. Abnormal pulsation in connexion with the carotid, and less often in scapular and internal mammary arteries, should be looked for.

Palpation

The position of the apex beat, defined as the lowest and outermost part of the cardiac impulse where the palpating finger is uplifted, should be determined in that it is the most reliable clinical index of cardiac enlargement or displacement. The extent and character of the apical pulsation is then judged and a record made as to whether it is localized or diffuse, quiet or forcible. In the case of triple rhythm of hypertensive heart failure a characteristic wavy pulsation may be felt. In the rare examples of conspicuous aneurysmal dilatation of the lower moiety of the descending thoracic aorta, the heart is jerked forward against the chest during each systole. A thrill should be sought in the mitral area, over the base of the heart, and in the neck above the sternum and right clavicle. A thrill in the mitral area need not be timed in relation to the cardiac cycle because if aortic stenosis is absent it indicates mitral stenosis whether it is presystolic, systolic, or diastolic. A systolic thrill in the fourth left intercostal space, near the lateral border of the sternum, is present in a patient with a defect of the ventricular septum. Thrills in the pulmonary area are always systolic in time and connected with pulmonary stenosis or patent ductus arteriosus, and less commonly in auricular septal defect. In the aortic area a thrill indicates aortic stenosis and even when it happens in diastole, aortic stenosis will be found accompanying aortic incompetence. A systolic thrill in the neck has no significance in young subjects unless it is long and accompanied by a systolic murmur in the aortic area.

Assuming that the electrocardiogram has been taken, it is necessary to consider its interpretation. The written waves have been arbitrarily designated P, Q, R, S, T and U, representing the electrical changes taking place during the progress of cardiac contraction. The P wave is held to represent the electrical force produced by activation of the auricular muscle. The QRS and T waves are held to represent the electrical force generated when the ventricular muscle contracts and relaxes. The U wave is less well understood, but it probably represents

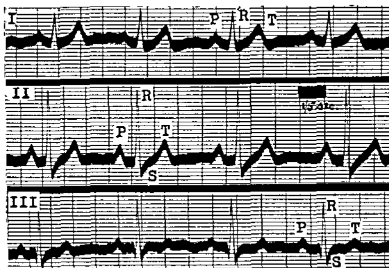


FIG 1.—Physiological electrocardiogram

some sort of re-adjustment of the electrical potential within the ventricle. The individual components of the QRS complex vary in number from subject to subject and from lead to lead The earliest QRS deflection which lies above the isoelectric level should be labelled R. Any downward deflection which precedes R, so defined, should be labelled Q. The first downward deflection following R should be labelled S The first upward deflection which may follow S should be labelled R′ and if any downward deflection follows R′ it is designated S′ The term "diphasic T wave" is applied to a double deflection of the T, one on each side of the isoelectric level; if the first deflection lies below the level, the diphasic T is of the minus-plus (∓) type, or if the reverse applies it is of the plus-minus (±) type

In the actual reading of an electrocardiogram attention should be paid in each lead to some six particulars, and a methodical analysis of these will show how the record differs from the normal, providing a diagnosis of the condition. The following are the six points and each in turn should receive separate consideration

1. **The rate.**
2. **The rhythm.**
3. **The length of the P–R period.**
4. **The direction of the electrical axis.**

that James Mackenzie was able to add so materially to our knowledge of cardiac arrhythmia. Nowadays, however, the polygraph has been largely superseded in the routine clinical examination of the heart by the electrocardiograph.

Electrocardiography

Electrocardiography provides a valuable aid in the recognition of many cardiovascular disorders, especially of coronary disease, conducting bundle branch injury, pericardial disease, and in the interpretation of obscure arrhythmia. The electrocardiogram should never be used by itself to decide diagnosis or prognosis, and any abnormality it may show is to be evaluated in conjunction with the clinical findings. Several types of electrocardiographs are now in use. They are described fully in the instruction booklets issued by the firms which supply the respective instruments, and the technique of recording electrocardiograms is best acquired from a study of these manuals. The two electrodes taking off the electrical current consequent on cardiac action may be placed at different points of the body. Convention has acknowledged the value of three limb leads, but agreement on the best chest leads to use is not yet universal Table I details the location of the electrodes in the leads which are referred to in this book. The respective merits of unipolar leads, CR leads, and CF leads, in the diagnosis of cardiovascular disease are still being considered, and only the CR leads are considered in this book, it can be said already that the other two kinds are unlikely to prove superior in clinical electrocardiography for the changes in the one are also evident in the other, and often to better advantage.

TABLE I

Showing the position of the electrodes in the limb and three chest leads described in this book C stands for chest, R for right arm, and the numbers 1 to 7 for the station of the exploring electrode. (Chest leads with stations 2 to 6 are not described here, apart from CR_4, i.e., IVR in the healthy subject.)

Designation of leads		*Position of electrodes*	
		Indifferent electrode	*Exploring electrode*
Limb	I	Right arm	Left arm
,,	II	Right arm	Left leg
,,	III	Left arm	Left leg
Right pectoral	CR_1	Right arm	At right border of sternum in fourth intercostal space
Apical	CR_4	Right arm	Outer side of apex beat
Apical	IVR	Right arm	Outer side of displaced apex beat
Posterior axillary	CR_7	Right arm	Left posterior axillary line at level of apex beat

5. The direction, amplitude and form of the P wave, QRS complex, and the T wave.
6. The form of the R–T segment.

A normal or physiological electrocardiogram (Fig. 1) should conform to the following requirements in respect of these six desiderata.

1. The rate is neither too slow nor too rapid (60 to 100 per minute).
2. The rhythm is normal, the impulse arising in the sinoauricular node (sinus rhythm).
3. The P–R period is neither too short nor prolonged (0·10 to 0·22 sec).
4. There is no abnormal deviation of the electrical axis.
5. The P wave, QRS complex, and the T wave, are upright in the limb leads and normal in amplitude and form
6. There is no abnormal deviation of the R–T segment.

When the excitation wave spreads through the heart it is attended by a wave of electrical activity which takes a complicated path Its diffuse course may be

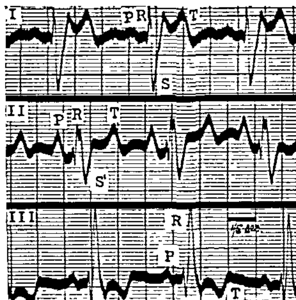


Fig. 4—Right electrical axis deviation

represented by an axis, the projection of which is determined in relation to a triangle constructed by the orthodox limb leads of the electrocardiogram. The resultant of the projection of the true axis of the disturbed electrical potential from the heart contraction is the electrical axis of the electrocardiogram. If this electrical axis is not deviated from the normal, the main deflection of the QRS complex is upright in each of the three limb leads (Fig. 2)

In *left electrical axis deviation* (Fig. 3), the main deflection of the QRS complex in lead III is downward and the R wave in lead I is taller than the R wave in lead II.

These changes are found in preponderant enlargement of the left ventricle as in hypertension and aortic incompetence. It is also a feature of left bundle branch block and is sometimes caused by a displacement of the heart upwards and to the left. Occasionally it is present in the absence of any abnormality of the heart.

In *right electrical axis deviation* (Fig. 4) the main deflection of the QRS complex in lead I is downward and the R wave in lead III is taller than the R wave in lead II. These changes are found in preponderant enlargement of the right heart as in mitral stenosis, congenital heart disease, or sometimes in emphysema. It is also a feature of right bundle branch block. Occasionally it is found in healthy subjects.

Enough has been said at this stage to enable the reader to analyse electrocardiograms in accordance with the scheme which has been outlined, and other tracings will be shown in connexion with the disease which they illustrate.

Radiography

As it is now admitted that x-ray examination is a valuable aid to the diagnosis of cardiovascular disease it is no longer necessary to exalt its advantages over the *shortcomings of percussion as a means of discovering the limits of the heart.* Although the position of the apex beat gives some information on the size of the heart, this is never precise and it only deals with the width of the heart, and its depth can only be made out by cardioscopy (radiological examination of the heart and great vessels) in the orthodox oblique positions. Further, it provides the only way of knowing the shape of the heart, which applied to diagnosis is even more important than knowing the size. Examination of the cardiovascular system is, therefore, never complete without cardioscopy, and particularly is it of value in the following circumstances to tell the significance of a doubtful mitral systolic murmur , to give precision in the diagnosis of congenital heart disease , to help in the early recognition of aortitis, mitral stenosis, and cardiac aneurysm , to detect one of the first objective symptoms of heart failure, namely, congestion of the hilar vessels ; to judge progress during treatment in any case of heart failure by noticing from time to time the extent of the pulmonary congestion , to estimate prognosis in relation to the signs of failure and the degree of cardiac enlargement.

X-ray examination of the heart may be applied in three different ways (Fig. 5), and a short description of these will now be given.

Orthodiagraphy.—By means of the orthodiagraph the exact outline and image of the heart can be recorded. Parallel x-rays are obtained by using a narrow beam projected from the x-ray tube which is moved so that the beam traces the cardiac outline synchronously with a greased pencil which writes in the centre of the spot of light on the fluorescent screen. The outline of the thoracic wall is similarly traced. The orthodiagram depicts the true size and shape of the heart, and therefore any measurements applied to different moieties of the heart and greater vessels should be determined by this method The tracing thus obtained does not, however, represent to the observer as true an impression of the thoracic structures as that conveyed by a teleradiogram of the chest.

x--y A

x--y B

x--y C

Fig 5.—Diagrams to illustrate the relative distortion of the heart shadow taking place during the x-ray examination of the heart by orthodiagraphy (A), teleradiography (B), and cardioscopy (C), x-y is the width of the projected heart shadow, in (A) it is real, but it is exaggerated in (B) and especially in (C)



Fig 6



Fig 7

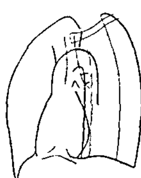


Fig 8.

Outline of structures seen during cardioscopy (*see* Table II) Fig 6 shows the anterior position, Fig 7 shows the right oblique position, Fig 8 shows the left oblique position

Teleradiography.—The teleradiogram is taken with the x-ray tube placed six feet away from the film At this distance the rays only diverge slightly so that the distortion, inseparable from a method in which the rays diverge, is thereby largely obviated. Thus, a teleradiogram provides a substantially accurate record of the size and shape of the heart

Cardioscopy.—Although this method involves examination of the heart and great blood vessels by divergent rays, supplying a distorted image, the advantages of the ease and simplicity with which it is applied make it the best method to adopt in routine work. The heart is viewed by cardioscopy in the three orthodox positions, anterior (patient facing screen), right oblique (patient turned half-left), and left oblique (patient turned half-right).

Normal cardiac outline.—The heart is seen as a flask-shaped silhouette with about one-third of the shadow to the right of the midline and two-thirds to the left. The structures to be identified from inspection in each of the three orthodox positions are listed below in order, from above downwards. Table II should be read in conjunction with the three diagrams shown in Figs. 6, 7 and 8.

During cardioscopy also, the progress of a thick suspension of barium down the oesophagus may be watched and this provides a valuable aid in the diagnosis of

TABLE II

Structures for identification by cardioscopy arranged from above downwards in the three orthodox positions

Position of patient	*Right border of shadow*	*Left border of shadow*
Anterior	Superior vena cava (Ascending aorta within) Right auricle Inferior vena cava	Aortic knuckle (Descending aortic arch) Pulmonary artery or appendage of the left auricle Left ventricle
Right oblique	Descending aorta Left auricle Inferior vena cava	Ascending aorta Pulmonary artery Left ventricle
Left oblique	Superior vena cava Ascending aorta Right ventricle	Aortic triangle Aortic arch Aortic window Left pulmonary artery Descending aorta Left ventricle

cardiovascular disease. In its thoracic course the oesophagus is impressed by four structures, the aortic arch, left bronchus, left auricle and descending aorta.

Abnormalities of these will disturb the position or contour of the natural oesophageal impressions (Figs. 9, 10 and 11). Of special importance is a deformed left auricle impression viewed in the oblique positions.

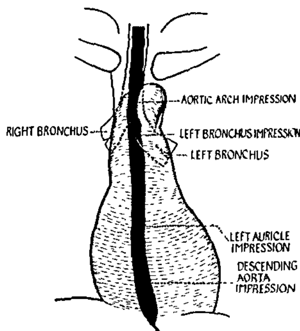


FIG 9.—Composite teleradiogram, showing barium-filled oesophagus superimposed on the cardiovascular silhouette in the anterior position, from a healthy subject aged 24 years

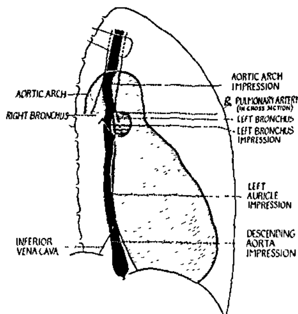


FIG 10.—Composite teleradiogram, showing barium-filled oesophagus superimposed on the cardiovascular silhouette in the right oblique position, from a healthy subject aged 24 years.

Kymography.—X-ray kymography is a means of recording the movements of the heart. A lead plate with horizontal slits spaced at regular intervals is interposed between the patient and the film Each slit allows a narrow beam of rays

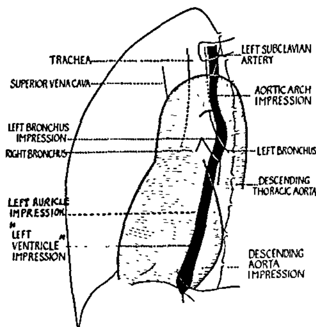


FIG 11 —Composite teleradiogram, showing barium-filled oesophagus superimposed on the cardiovascular silhouette in the left oblique position, from a healthy subject aged 24 years

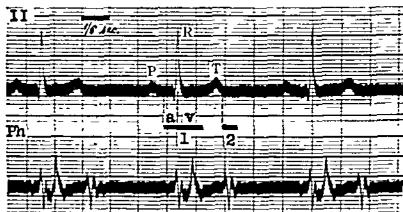


FIG. 12 —Phonocardiogram showing dual rhythm in a healthy subject aged 25 years In this and subsequent figures, Ph is the sound record, and a and v are the auricular and ventricular parts of the first heart sound respectively

to fall on the film. The record is taken either with the grid stationary and the film moving, or with the film stationary and the grid moving. The kymogram shows serrations along the cardiac outline created by excursions of the heart during systole and diastole. This method of investigation is not likely to become

routine procedure, although it can sometimes aid in the differential diagnosis of aortic aneurysm from a new growth, and it can help in the recognition of pericardial disease, and demonstrate a quiet area of the heart resulting from infarction or localized pulsation in early cardiac aneurysm.

Phonocardiography

The recording of heart sounds and murmurs is likely to become a routine test in clinical cardiology. It has already proved its worth in research directed to the more precise interpretation of certain auscultatory signs. Since a few phonocardiograms have been included in the text, it is necessary to describe the normal tracing (Fig. 12), which was obtained by a Cambridge string galvanometer in which a glass tube connecting the chest piece and amplifier measured 46 cm The auricular sound in such a tracing starts at the end of the P wave of the electrocardiogram and merges into the ventricular part of the first sound which starts near the S wave and never in front of the R wave. The second sound begins near the end of the T wave.

Cardiac catheterization

In this procedure a catheter is introduced by way of the antecubital vein and its tip is placed successively, under radiological observation, in the superior vena cava, the right auricle, the right ventricle, and the pulmonary artery. The pressure in these separate parts can be taken in different clinical conditions and the effect on the venous pressure of standard remedies may be tested. Estimation of the oxygen content of samples of blood from the different stations will help in the differential diagnosis of certain congenital defects if this has been difficult on clinical grounds, because a significant increase of the oxygen content in a particular chamber will indicate an entry of oxygenated blood at this site.

Other methods

Other aids are of value in the study of cardiovascular disorders, but they are only necessary in certain cases. The Wassermann reaction, if positive, may emphasize the syphilitic nature of aortic incompetence or a localized deformity of the aorta. A blood count will be useful in the diagnosis of anaemia, certain forms of congenital heart disease, or doubtful cardiac infarction, and a blood culture to give significance to certain cardiac murmurs. The blood urea is useful when cardiorenal disease is suspected. The basal metabolic rate sometimes helps to decide the influence of the thyroid gland in causing certain cardiovascular changes The erythrocyte sedimentation rate may often prove of value Oscillometric readings obtained by the Pachon's oscillometer or Tycos manometer contribute to the detailed investigation of a patient presenting ischaemia of a limb, but radio-arteriography is seldom necessary for the routine study of this condition. Estimation of the circulation time has a limited application in clinical diagnosis. Tomography may also help in the investigation of an obscure shadow connected with the heart and greater vessels.

CHAPTER 2

HEART SOUNDS AND MURMURS

HEART SOUNDS

VIEWS on the precise mechanism of the first and second heart sounds have long remained orthodox. Thus, it was thought that the first sound arose chiefly from the closing of the auriculo-ventricular valves, but phonocardiography nominates contraction of the auricles and ventricles as the important mechanism in its production. The second sound in the pulmonary area is caused chiefly by the closure of the pulmonary valve, and of the aortic valve in the aortic and mitral areas

THE SOUNDS IN DUAL HEART RHYTHM

Accentuation of the first heart sound

The first sound in the mitral area is often loud in health, especially if tachycardia is present It is a feature of mitral stenosis unless it is obscured by a systolic murmur. Accentuation of the first sound is also common in hypertension.

Accentuation of the second heart sound

The relative intensity of the second sound in the aortic and pulmonary areas varies with age, so that during childhood the pulmonary sound is louder than the aortic, in adults the sounds are expected to be of equal intensity, and in older subjects the aortic sound is a little louder than the pulmonary. Allowance for this natural age variation must be made before assigning to either sound an abnormal intensity. Hypertension is a common cause of a loud second sound in the aortic area, but the sound is normal in many cases. Should the sound be loud in the absence of hypertension preponderant atheroma of the aorta or aortitis might be present to account for it, but it is often found in health. Early aortic incompetence can be present when the second sound is loud, so that care is needed to search for the diastolic murmur in such patients. Although accentuation of the second sound in the pulmonary area results from those conditions in which the blood pressure within the pulmonary circulation is raised, it is a common sign in health. This auscultatory sign does not give much help in the diagnosis of mitral stenosis, admittedly it is often loud when the heart is moderately enlarged from mitral stenosis, but it is not loud in the majority of cases of early mitral disease, while it is frequently loud in healthy young subjects. Congenital heart disease, emphysema, and thyroid toxaemia, are other conditions which might accentuate the sound. A loud second sound in the mitral area is the outcome of the same conditions which increase the intensity of the sound in the aortic area.

Splitting of the mitral first sound

The first sound, made up of its auricular and ventricular components, is never doubled or reduplicated. If the sound appears to display a duplicity it is the result of either slight separation of the two natural components, or the accentuation of one over the other. Thus, the mechanism is a form of splitting and no additional sound has been introduced

The importance of this auscultatory sign lies in the frequency with which it is mistaken for the presystolic murmur of mitral stenosis It differs from this,

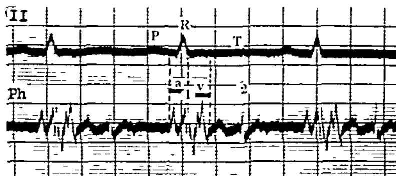


FIG. 13 —Splitting of the first heart sound The auricular sound is prominent (auricular type)

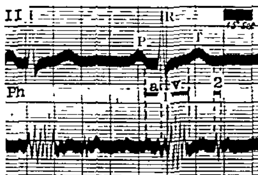


FIG. 14 —Splitting of the first heart sound The ventricular sound is prominent (ventricular type).

however, in that the sound might be represented as "r-rup" and not "thur-rup," it is better heard towards the midline and in the upright posture, and there is no evidence of heart disease It is met with frequently and in three circumstances.

1. *In health.*—Splitting of the first heart sound in the mitral area is found commonly in healthy thin subjects at all ages. It is heard to best advantage near the xiphisternum and in the upright posture. No sign of heart disease can be identified with this auscultatory finding on clinical or radiological examination. It is seldom absent over a depressed sternum The electrocardiogram is normal and the P–R period is not prolonged. The phonocardiogram may be distinctive and showing either an exaggerated auricular sound wave, the *auricular* type (Fig. 13), or ventricular wave, the *ventricular* variety (Fig. 14). The splitting

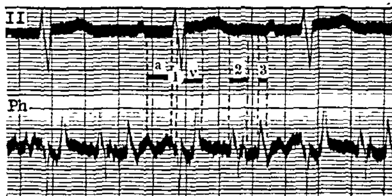


FIG 15.—Splitting of the first heart sound in a healthy subject showing slight lengthening of the P–R period

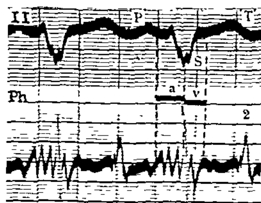


FIG 16.—Splitting of the first heart sound in bundle branch block. R–S period is prolonged so that ventricular part of first sound is delayed

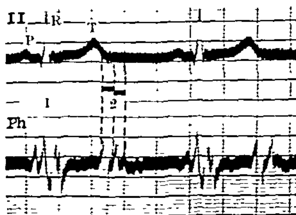


FIG 17.—Splitting of the second heart sound at the pulmonary area in a case of mitral stenosis

shown by patients with hypertension also conforms with one of these two varieties identified with health.

2. *In lengthened P–R period*—It is natural to expect that moderate separation of the auricular and ventricular components of the first heart sound as the outcome of delay in the auriculo-ventricular conduction of the heart impulse, will give rise to splitting on auscultation (Fig. 15). A greater separation would produce two distinct sounds initiating triple rhythm. As the P–R period which causes splitting is only a little in excess of 0·2 second, any subject presenting this sign should not on this count alone be considered unhealthy.

3 *In bundle branch block.*—Corresponding with the lengthening of the R–S period in the electrocardiogram of bundle branch block, is the delay in the appearance of the ventricular part of the first heart sound, and this causes splitting of the sound (Fig 16). Since the P–R period is also commonly prolonged in bundle branch block, the common incidence of splitting of the first heart sound in this condition is readily understood. Indeed, this auscultatory sign in elderly subjects should direct attention to the possibility of a lesion of the bundle, and an electrocardiogram should become a routine test in such cases.

Splitting of the pulmonary second sound

When the second sound in the pulmonary area is accentuated it usually demonstrates splitting as well (Fig. 17) It may be caused by the pulmonary valve closing before the aortic, but although it is commonly found in conditions associated with a raised blood pressure in the pulmonary circulation, notably mitral stenosis with moderate enlargement of the heart, and congenital heart disease, it is often present in healthy young subjects. The sign is without great significance unless it is the result of asynchronous closure of the semilunar valves in bundle branch block.

Distant heart sounds

No great reliance should be placed on an indistinct first sound in the mitral area as a sign of disease, because it may be distant in healthy elderly subjects and especially in those with a thick chest wall or emphysema ; the sign is often found in hypertensive heart failure, cardiac infarction, and pericardial effusion, but care should be taken not to accept it as evidence of heart disease in the absence of other supporting signs.

A distant second sound in the mitral area may mean that an early aortic diastolic murmur is present and this should be tested in other areas, although mention has already been made of the common association of a loud aortic second sound with an early diastolic murmur.

THE SOUNDS IN TRIPLE HEART RHYTHM

By triple rhythm is meant the cadence produced when three separate sounds recur in succeeding cardiac cycles. It is a common auscultatory sign, and in patients referred for examination of the heart at least, it is almost as common as the familiar dual rhythm produced by the first and second heart sounds. The appreciation of this common incidence of triple rhythm will come to us as soon as we adopt during clinical auscultation the simple form of self-catechism, "*Do I hear more than two heart sounds?*"

A clinical classification of triple rhythm should deal with its cause in terms of heart disease, and should deliberately exclude conjecture concerning the actual mechanism of the added sound. When the position of the added heart sound in the cardiac cycle is considered alongside the clinical state in patients with triple rhythm it is possible to place them in three groups (Fig. 18 and Table III). As a rule, unless under the handicap of tachycardia, the position of the extra

		Systole	DIASTOLE		Systole	
DUAL RHYTHM		lup – – – dup 1 2	. . . .		lup . . . dup 1 2	. . .
TRIPLE RHYTHM	.	lup – – – dup 1 2	dub 3		lup – – – dup 1 2	dub 3
	lub 4	lup – – – dup 1 2	. . .	lub 4	lup – – – dup 1 2	. .
	.	lup - dub dup 1 S 2	. . . . .		lup - dub dup 1 S 2	.
QUADRUPLE RHYTHM	lub 4	lup – – – dup 1 2	dub . . 3	lub 4	lup – – – dup 1 2	dub 3

FIG 18 —Showing position of heart sounds in cardiac cycle in different types of triple rhythm S—systolic extra sound Fourth heart sound occurs in *ventricular* diastole but in *auricular* systole

sound can be told by auscultation, aided by the clinical data, and without phonocardiography, although the test is necessary for the interpretation of a difficult case The three kinds of triple heart rhythm will now be described.

Addition of the third heart sound

In this group the added sound appears early in diastole and is the third heart sound. The auscultatory sign is found in young healthy subjects, or in right heart failure. Recognition of each kind comes from a regard of the site of maximal intensity of the sound, the effect of posture upon it, and the presence or absence of heart disease.

The third heart sound in health.—This innocent kind of triple rhythm (Fig. 19) is best heard over or a little internal to the apex beat in the reclining posture, and it usually becomes inaudible in the upright posture unless there is tachycardia causing it to persist ; it is found in young healthy subjects and is common in

TABLE III. Comparing the characteristics of the three different types of triple heart rhythm

Kinds of triple rhythm	*Addition of the third heart sound*		*Addition of the fourth heart sound*		*Addition of a sound in late systole*
Place of the added sound in cardiac cycle					
Cause	In health	*In right ventricular failure* Mitral stenosis Hypertension Congenital heart disease Thyroid toxaemia Emphysema, primary pulmonary hypertension, pulmonary embolism Anaemia Constrictive pericarditis Cardiac infarction Familial cardiomegaly	In delayed A-V conduction	*In left ventricular failure* Hypertension Aortic incompetence Cardiac infarction	In health
Site of maximal intensity	Internal to mitral area	Over displaced apex beat, at xiphisternum	Internal to mitral area	At xiphisternum, over displaced apex beat	Internal to mitral area
Effect of erect posture	Usually disappears	Persists	Persists	Persists	Persists
Effect of auricular fibrillation	None	None	Disappears	Disappears	None
Radiological features	Pulmonary artery full	Right heart often enlarged	None	Left heart enlarged pulmonary congestion	None
Probable mechanism	Vibration of ventricular wall from inrush of blood in early diastole		Auricular contraction	Vibration of hypotonic ventricle from auricular systole	Not known
Special features	In young subjects, never after 40	Sound is followed by murmur in mitral stenosis	Usual cause of triple rhythm in bundle branch block	Double systolic impulse felt	Comparable with the innocent murmur in late systole

children and youths, much less common at the age of 25, and is never heard after 40 years of age If the third heart sound, elicited by auscultation in a subject over 40 years of age, is ever regarded as incidental, the deduction should not be made lightly, and only when the electrocardiogram shows no elongation of the P–R period (with or without bundle branch block), when cardioscopy shows the absence of cardiac enlargement (minimal in heart failure from cardiac infarction, emphysema, constrictive pericarditis, and thyroid toxaemia), and when the phonocardiograph has demonstrated an obvious third heart sound. When such rules are made to apply, it is likely that very few, if any instances of triple heart rhythm from

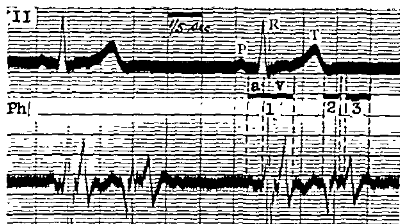


Fig 19 —Triple rhythm from addition of the third heart sound From a healthy male, aged 21 years.

the addition of the third heart sound will be found in healthy subjects over the age of 40 years It is this truth which invests such importance in this auscultatory sign. On cardioscopy, slight prominence of the pulmonary artery, prominence of the right auricle, and squat appearance of the heart are sometimes present (Fig. 20)—appearances in keeping with the common incidence of this auscultatory sign in young subjects in whom similar features are common to the normal teleradiogram (Fig. 21), but this radiological pattern is sometimes wanting in this innocent form of triple rhythm.

The third heart sound in right ventricular failure.—Here the third heart sound is loudest, as a rule, over the displaced apex beat , it persists with the patient in the upright posture, and there is evidence of disease which has caused enlargement or failure of the right heart such as mitral stenosis, hypertension, thyroid toxaemia, congenital heart disease, emphysema, pulmonary embolism, primary pulmonary hypertension, constrictive pericarditis, anaemia, cardiac infarction, or some rare forms of cardiomegaly. This kind of triple rhythm is not limited to young subjects like the innocent variety, and it bears repeating that a third heart sound in a patient over 40 years of age indicates right heart failure, and one of the enumerated causes should be sought to account for it.

This triple rhythm is present in more than half of the number of patients with *mitral stenosis* (Fig. 22), and is uninfluenced by the addition of auricular

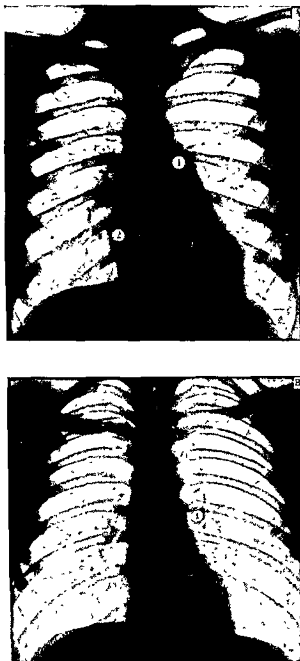


FIG 20 —Triple rhythm from addition of the third heart sound Teleradiograms in two healthy subjects aged 23 years (A) Prominence of pulmonary artery (1) and the right auricle (2) with triple rhythm, and (B) good pulmonary bay (1) with dual rhythm

fibrillation. Usually, the third heart sound in mitral stenosis is obscured by a mid-diastolic murmur.

When the left ventricle fails in *hypertension* a triple rhythm due to the presence

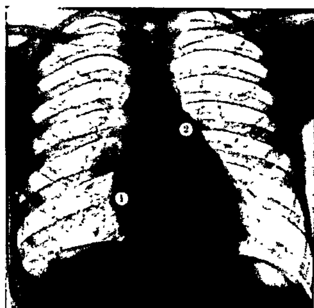


Fig. 21—Characteristic heart outline in healthy child aged 12 years. There is prominence of the right auricle (1) and pulmonary artery (2)

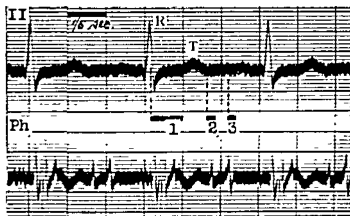


Fig. 22—Triple rhythm from addition of the third heart sound in a female aged 28 years with mitral stenosis

of the fourth heart sound is commonplace, but when the right heart has failed it is the third heart sound which creates the triple rhythm Although the former variety may give way to the latter as the disease progresses, the triple rhythm initiated by the addition of the third heart sound might be the first to appear in

many patients with hypertensive heart failure. The rhythm may disappear on auscultation for a time during treatment with rest and mercurial diuretics

Triple rhythm in *thyroid toxaemia* due to the addition of the third heart sound means that enlargement of the right heart and pulmonary artery has been a complication of the condition. No cardiac enlargement is present in those cases with thyroid disease which show dual rhythm.

Few cases of *congenital heart disease* show triple rhythm, but in those that do, great enlargement of the right heart is a feature, and it is especially common in auricular septal defect.

Clinical heart failure with conspicuous enlargement of the right heart is rare in *emphysema*, but such changes can be presumed present if this kind of triple rhythm is found. The finding of this auscultatory sign is an important event in such a case because it signifies that the patient cannot survive many months The same triple rhythm may be heard in the uncommon condition known as *primary pulmonary hypertension*. Triple rhythm from addition of the third heart sound is a valuable supporting sign in a patient taken suddenly ill with severe shortness of breath which suggests the diagnosis of *pulmonary embolism* Indeed, it supplies the most reliable clue in the recognition of this critical illness, short of electrocardiography. A third heart sound is also an invariable finding in *constrictive pericarditis*, and it is present in *anaemia* when there is enlargement of the heart. *Cardiac infarction* can induce either kind of triple rhythm due to the addition of either third or fourth heart sounds and the former is best heard over the xiphisternum.

Addition of the fourth heart sound

Since the adventitious sound in this form of triple rhythm occupies a place later than that occupied by the third heart sound, it is called the fourth heart sound, and it immediately precedes the first heart sound It only precedes, however, the *ventricular* moiety of the first sound because it occurs during auricular systole. As in the case of triple rhythm from the addition of the third heart sound, so also in this variety there are two classes; in the first the supernumerary sound is produced by the auricular systole and appears only when auriculo-ventricular conduction is delayed; in the second, although the sound is produced in or by the left ventricle affected by failure, regulated contraction of the auricle is necessary for its production, and it is never heard in auricular fibrillation

The fourth heart sound in delayed A–V conduction.—In healthy subjects the sound produced by auricular systole is easily recorded by the phonocardiograph, but its proximity to the sound of ventricular systole hinders the appreciation by clinical auscultation of these separate moieties of the first heart sound. When A–V conduction is delayed and the P–R period of the electrocardiogram is prolonged to upwards of 0 25 second, it is usually possible, especially in young subjects, to hear both sounds, creating a triple rhythm (Fig. 23). This triple rhythm, best heard a little internal to the mitral area and persisting in the upright posture, does not by itself tell of heart disease because some 50 per cent of the subjects presenting it can be counted as healthy.

The fourth heart sound in left ventricular failure.—*Hypertension* is the usual cause of left ventricular failure initiating this form of triple rhythm (Fig 24); it is only met with in a few cases of *aortic incompetence.* So far I have not

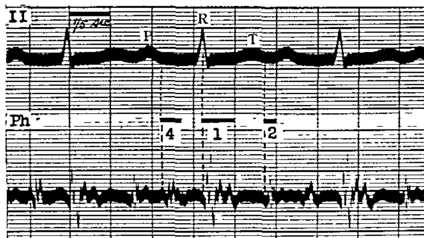


Fig 23.—Triple rhythm from addition of the fourth heart sound in a female aged 25, who did not show any evidence of heart disease apart from delayed A–V conduction

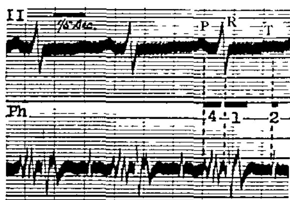


Fig 24.—Triple rhythm from addition of the fourth heart sound in a male aged 69 years, with hypertensive heart failure

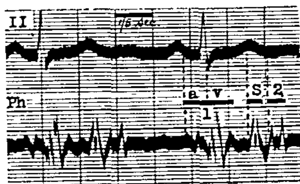


Fig 25.—Triple rhythm from addition of a sound (S) in late systole in a healthy male aged 37.

met it in aortic stenosis. *Cardiac infarction* can initiate triple rhythm on its own, but often it produces it by precipitating heart failure in hypertension. The abnormal rhythm from addition of the fourth heart sound in left ventricular failure is sometimes best appreciated over the displaced apex beat, but as a rule it is best heard near the xiphisternum. A characteristic dual impulse is often felt over the cardiac area in these patients and especially when the supernumerary sound is well heard; this is best appreciated by placing the naked ear to the chest when the impulse can be felt and the sound heard at the same time. Tachycardia is common in such cases and it makes the rhythm more audible. In view of the paucity of physical signs in left ventricular failure, this auscultatory sign has great value, for its presence calls for the early use of mercurial diuretics and digitalis.

An extra heart sound in late systole

This third kind of triple rhythm is likely to be mistaken for the first variety, but careful auscultation should readily localize the supernumerary sound *in front of* and not *after* the second sound (Fig 25). This auscultatory sign is uncommon, but when found it is not evidence of heart disease, for the subjects presenting it are healthy. Its position in systole corresponds with that of the *late systolic murmur* which is also an innocent finding.

Triple rhythm is a common auscultatory sign, and it can be of great aid in the diagnosis of a cardiovascular disorder. It should be sought specifically in every case, and when found, the supernumerary sound in the cardiac cycle should be traced and its significance determined in the light of clinical findings and in accordance with a classification such as that which is proposed here.

THE SOUNDS IN QUADRUPLE HEART RHYTHM

Four heart sounds in each succeeding cardiac cycle is not an unusual event, and may result from the combination of a prolonged P–R period initiating the fourth

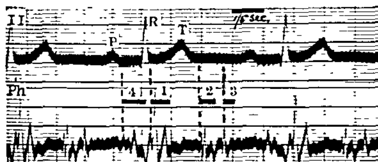


FIG 26—Quadruple rhythm Addition of the third heart sound in mitral stenosis and of the fourth heart sound from delayed A–V conduction

heart sound, and the addition of the third heart sound from right heart failure as, for instance, in mitral stenosis (Fig. 26).

HEART MURMURS

All murmurs have a meaning, and it is necessary to apportion to each its significance in terms of disease, in or outside the heart, or of altered function.

The interpretation of murmurs in the past has not been altogether precise, but since the introduction of cardioscopy and phonocardiography as accessory methods of examination, heart murmurs have been accorded a value in diagnosis which takes premier place amongst all the objective symptoms of cardiovascular disease. Indeed, care in eliciting the exact physical characteristics of murmurs gives every clinician a means of interpreting precisely the nature of most of the presenting clinical problems. The importance of examining the distinctive features which may identify a murmur with a particular cardiac disorder is, therefore, obvious, and it is now proposed to enumerate these in the case of all systolic and diastolic murmurs in the areas recognized for routine auscultation

SYSTOLIC MURMUR IN THE MITRAL AREA

Mitral stenosis

It is the custom in this book to regard mitral stenosis as a comprehensive disease and not solely as a condition producing a gradual closing effect of the mitral valve. Thus, a disease caused by rheumatic fever is envisaged, in which a murmur in systole or mid-diastole is heard on auscultation, in which enlargement of the left auricle and the right heart can be made out at cardioscopy, and in which auricular fibrillation and heart failure are common events responding well to digitalis, and in which intracardiac thrombosis and embolism are also common complications That is why it is held here that whenever a systolic murmur in the mitral area is regarded as evidence of mitral disease it should be referred to as mitral stenosis and not mitral incompetence to which reference will be made later.

The systolic murmur of mitral stenosis is rough in character ; it is moderately long and easily heard so that although it might diminish in intensity during deep inspiration it is never annulled by this manoeuvre. Although always audible in the erect position, it is better heard in the reclining posture and especially when the patient is inclined on the left side Occasionally in this position the murmur changes into a presystolic murmur following tachycardia induced by exercise A thrill may accompany the murmur. Short of these last two characteristics, this systolic murmur cannot by itself indicate mitral stenosis, although the assumption is justified after hypertension, aortic incompetence or stenosis, and congenital heart disease have been excluded by clinical examination. A mid-diastolic murmur should be sought in every case in which a systolic murmur is thought to arise from mitral disease, for it is commonly present.

The murmur is distinctive in the phonocardiogram (Figs. 27, 28 and 29), for *it commences earlier than other systolic murmurs* ; it usually starts in auricular systole and before the S line (a line drawn through the end of the S wave of the electrocardiogram to meet the phonocardiogram) and a mid-diastolic murmur is an invariable finding.

Aortic incompetence

A systolic murmur in the mitral area is a feature of all, even early, cases of aortic incompetence. If the nature of the aortic lesion is rheumatic, the possibility of associated mitral stenosis should be considered, although a systolic mitral murmur in this circumstance cannot be regarded as evidence of mitral disease, seeing that it is expected in patients with non-rheumatic aortic incompetence. The murmur usually commences at the S line in the phonocardiogram or soon after.

Aortic stenosis

A systolic murmur in the mitral area is expected in aortic stenosis and in some cases the murmur is as loud as the one heard in the aortic area, it usually commences at the S line in the phonocardiogram or soon after

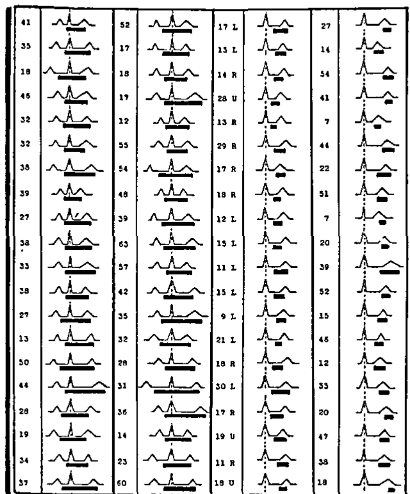


FIG 27 —The position of mitral systolic murmurs (represented by black lines) in relation to the electrocardiogram in 40 patients with mitral stenosis (20 with a presystolic and 20 with a systolic murmur), and in 40 healthy subjects with innocent murmurs (20 with the murmur in mid-systole and 20 in late systole) Figures denote ages Letters in the third column designate the clinical classification for innocent murmurs, thus, R is the murmur of Reclining Posture, U the murmur of Upright Posture, and L the Loud Variety.

Hypertension

A mitral systolic murmur is common in hypertension whenever much cardiac enlargement is present. This accounts for the error in diagnosing mitral stenosis in patients with hypertension as the only obvious abnormality. The murmur starts in the mid-systole and later than the S line in the phonocardiogram.

Cardiac aneurysm

When aneurysm of the heart following cardiac infarction has given rise to moderate cardiac enlargement, a mitral systolic murmur is expected.

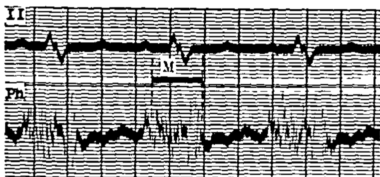


FIG 28 —Auricular systolic murmur in mitral stenosis in which auscultation showed a systolic murmur. The auricular murmur is continued from the mid-diastolic murmur

Heart block

A systolic murmur in the mitral area is common in heart block whenever much cardiac enlargement is present

Conducted murmurs

It is common to hear in the mitral area a murmur which has its maximal intensity in one of the other cardiac areas. The systolic murmurs of pulmonary stenosis,

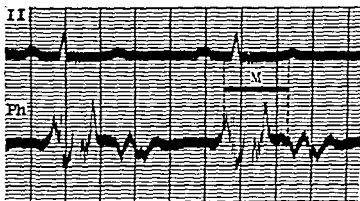


FIG 29.—Auricular systolic murmur in mitral stenosis in which auscultation showed a systolic murmur

patent ductus arteriosus, defect of the ventricular or auricular septa, and coarctation of the aorta, are examples of this.

Haemic murmur

This murmur has a soft or roughish quality, and when heard in the mitral area it is usually heard in the pulmonary area as well. It becomes less loud and it

may even disappear when the patient assumes the upright posture It also disappears when the anaemia is relieved. Its position in the cardiac cycle is in mid-systole.

Fever and tachycardia

Tachycardia from thyroid toxaemia or from any other cause, and particularly if it is accompanied by fever, often produces a systolic murmur in the mitral area. The murmur has no significance in prognosis and it disappears when the fever abates and the tachycardia subsides

Cardiac displacement

It is common to find a systolic murmur in the mitral area when the heart is displaced for some distance from any cause such as pulmonary fibrosis, pneumothorax or hydrothorax If the displacement is temporary the murmur disappears when the normal position of the heart is restored.

THE INNOCENT MITRAL SYSTOLIC MURMUR

The clinical diagnosis of an innocent murmur is not to be lightly undertaken, although in practice less harm might be done by failing to discover some cases of early mitral disease than to commit to a life of unwarranted invalidism a healthy young subject presenting an innocent murmur. Indeed, close attention to the characteristics of this murmur is long overdue. From the clinical and cardiographic examination of healthy subjects showing an innocent murmur it has been possible to describe this murmur for five clinical groups of patients The fact that the mechanism of the innocent murmur has evaded an explanation must not encourage conjecture on its ways of production, and above all it must not be allowed to impede the consolidation of a clinical syndrome which will by itself decide the innocent nature of the murmur.

TABLE IV

Distinctive clinical features in 330 healthy subjects with an innocent murmur

Special features	*The murmur in mid-systole* (262)				*The murmur in late systole* (68)
	The murmur of reclining posture (135)	*The murmur of upright posture* (80)	*The loud variety of murmur* (7)	*The parasternal murmur* (40)	
Age	Young subjects	Young adults	Young subjects	Older adults	Any age
Character	Blowing	Blowing	Blowing	Blowing	Blowing
Intensity	Not loud	Not loud	Loud	Loud	Loud
Effect of deep inspiration on intensity	Disappears to auscultation as a rule	Disappears to auscultation as a rule	Persists	Persists	Persists
Effect of posture on intensity	Louder in reclining posture	Louder in upright posture	Trivial	Trivial	Trivial
Effect of posture on distribution	Murmur appears in pulmonary area on reclining	None	Towards axilla in upright, and towards sternum in reclining posture	None	None

The murmur in mid-systole

Apart from its selective position in systole (Fig. 27), which gives to this innocent *murmur its distinctive* character, there are other clinical features which helped to decide the innocent nature of the murmur short of its graphical representation. From a consideration of the site and intensity of the murmur and the influence *upon it of posture, it is possible to allocate the* cases in four clinical groups (Table IV)

The murmur of reclining posture—This murmur is roughish or blowing in character and is neither loud nor long, so that it often disappears on deep inspiration. It is best heard a little internal to the mitral area and is louder in

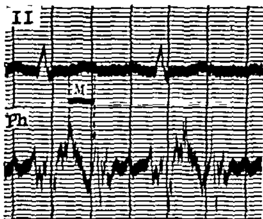


Fig 30—The innocent systolic murmur of reclining posture

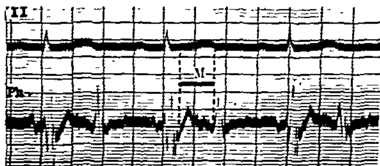


Fig 31—The innocent systolic murmur of upright posture

the reclining posture, when a murmur develops in the pulmonary area as well. Sometimes the murmur is audible in the pulmonary area in the upright posture, and in this event in the reclining posture the pulmonary murmur is louder than *the mitral. It is confined to younger subjects and is never met with after 40 years* of age.

In the phonocardiogram (Fig. 30) the murmur commences some way after *the S line and it often finishes before the second heart sound.*

The murmur of upright posture.—This murmur is roughish or blowing in character and is neither loud nor long, so that it often disappears on deep inspiration. It is best heard a little internal to the mitral area and is louder in the upright posture It is confined to younger subjects and it is seldom met with after 40 years of age.

In the phonocardiogram (Fig. 31) the murmur commences some way after the S line.

The loud variety—This murmur is roughish, whiffy or blowing in character and is loud, so that it seldom disappears on deep inspiration. In the reclining

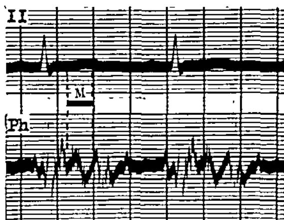


FIG. 32—The loud variety of innocent systolic murmur.

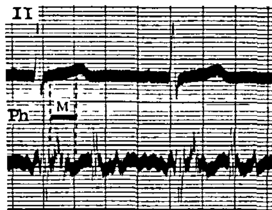


FIG 33—The innocent parasternal murmur

posture the murmur reaches upwards from the mitral area towards the sternum, whereas in the upright posture it is heard towards the axilla. It is confined to young subjects and it needs care to distinguish it from the systolic murmur of mitral disease.

In the phonocardiogram (Fig 32) the murmur commences beyond the S line.

The parasternal murmur—This murmur is blowing in character and is loud. It is loudest in the fourth intercostal space at the left lateral border of the sternum, although it is audible for some distance internal to this area. Unlike the murmur of ventricular septal defect, it is not associated with a thrill, or with cardiographic or cardioscopic abnormalities. This murmur is heard in older adults as well as in young subjects.

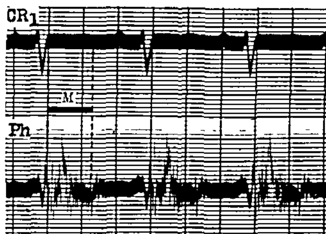


Fig. 34—The murmur of ventricular septal defect

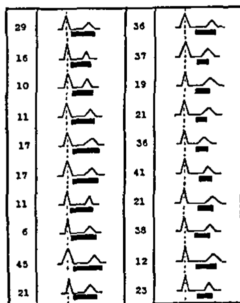


Fig 35—Position of murmurs (black lines) in relation to the electrocardiogram in 10 patients with ventricular septal defect, and in 10 subjects with an innocent parasternal murmur Numerals *denote ages*

In the phonocardiogram (Fig. 33) the murmur is in mid-systole, so that it commences beyond the S line, and in this way it differs from the murmur of ventricular septal defect, which is earlier, commencing at the S line (Figs. 34 and 35).

The murmur in late systole

This murmur is blowing in character and is loud so that it seldom disappears on deep inspiration; it is heard at all ages and its importance lies in the frequency with which it is mistaken for the murmur of mitral disease.

In the phonocardiogram (Fig. 27) the murmur is placed late in systole and starts near the commencement of the T wave of the electrocardiogram (Fig 36).

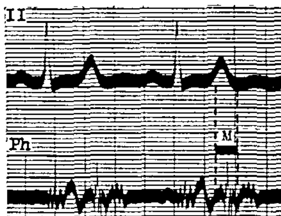


FIG. 36 —The innocent murmur in late systole.

An account of mitral systolic murmurs is incomplete without a statement on *mitral incompetence*. Before a disease can claim a place as a separate entity in clinical medicine it must present a consistent history in regard to aetiology, subjective and objective physical signs, and ultimately, pathological findings Thus, when a diagnosis of aortic incompetence is entertained, we envisage on clinical examination, a collapsing pulse, raised pulse pressure, increased pulsation in the neck, outward displacement of the apex beat, and an aortic diastolic murmur, at cardioscopy a distended left ventricle and increased aortic pulsation will be evident, and at necropsy the left ventricle will be found enlarged together with deformity from disease of the aortic valve No such constant pattern, either clinical, radiological, or pathological can be identified with mitral incompetence Indeed, a systolic murmur in the mitral area appears to be the only sign apportioned to it, and this has never been described as distinctive from the systolic mitral murmur common to many pathological states, notably mitral stenosis, aortic incompetence, aortic stenosis, and hypertension Is the mitral valve incompetent in these conditions? In order to answer this question there must be set up certain criteria which substantiate the presence of mitral incompetence To say that a systolic murmur signifies that blood regurgitates through the mitral opening is pure conjecture and cannot be admitted as evidence The presence of three conditions, however, establishes a diagnosis of mitral incompetence They are. the finding of specific enlargement of the left auricle at cardioscopy, a murmur synchronous with ventricular contraction in the phonocardiogram, and mitral valve disease and/or dilatation of the mitral ring at necropsy The first criterion, namely, enlargement of the left auricle as an effect of mitral incompetence, is accepted from an analogy with the form of cardiac enlargement which takes place in auricular and ventricular septal defects, when the cavity receiving the abnormal blood flow during systole (right auricle in the former and right ventricle in the latter), is the part of the heart which enlarges. When these three signs were sought specifically in the five clinical states enumerated above, they were only found in patients with rheumatic disease of the mitral valve (mitral stenosis) Even in those cases of mitral disease which show only a systolic murmur on auscultation the great majority when examined by phonocardiography demonstrate an auricular systolic murmur, and even in the few in which the start of the murmur coincides with ventricular systole there is also present as a rule a mid-diastolic murmur as evidence of mitral stenosis. The advice to forego the use of the term *mitral incompetence* is given because, so far from helping in the diagnosis of heart disease, it has handicapped it. In its place the

clinician must decide on the cause of the systolic murmur in terms of aortic incompetence, aortic stenosis, hypertension or cardiac enlargement from heart block. Even in mitral stenosis the lesser effect of incompetence, admittedly present, does not merit specific mention in diagnosis in that the term *mitral stenosis* is a comprehensive one embracing all the effects of this heart disease caused by rheumatic fever.

DIASTOLIC MURMUR IN THE MITRAL AREA

Early diastolic murmur

The early diastolic murmur in the mitral area obscures the second sound and is caused by aortic incompetence, or, less commonly, by pulmonary incompetence in auricular septal defect or in mitral stenosis (Graham Steell murmur) Should the murmur be rough in character aortic stenosis is conjoined with aortic incompetence. Sometimes the murmur is imperfectly heard, but at other times it is readily elicited, permitting a diagnosis even of aortic valvular disease from clinical auscultation in the mitral area.

Mid-diastolic murmur

This murmur, pathognomonic of mitral stenosis, follows immediately on the third heart sound, and is rough in character, sounding like *durr-* Although this murmur is often readily heard when there is moderate enlargement of the heart in mitral stenosis and in the presence of auricular fibrillation, it should be *diligently sought in cases of early mitral disease. A phonocardiogram* will usually record it even though it might not be audible on auscultation. The murmur persists in the upright position although it is best heard with the patient inclining on the left and over the displaced apex beat A thrill may accompany it.

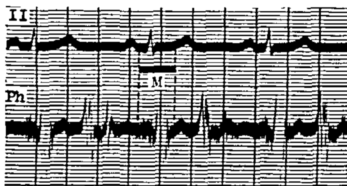


FIG 37—Auricular murmur in mitral stenosis in which auscultation showed a presystolic murmur

Presystolic murmur

This murmur is also pathognomonic of mitral stenosis. Both the terms diastolic and presystolic are misnomers for they apply only to the ventricle, and the murmur takes place during auricular systole (Figs 27 and 37) ; it disappears when auricular fibrillation sets in. The murmur is not crescendo in its graphic representation and this auditory impression is only created by the loud first heart sound which accompanies the auricular murmur ; should the P-R period be prolonged the murmur is

less likely to assume this characteristic. Although the presystolic murmur stands for mitral stenosis, other sounds simulate it so closely that care is needed in the interpretation. The student in medicine must make himself familiar with the characteristic sound " thur-rup," identified with a presystolic murmur, and be able to recognize it straightway. Indeed, it is sound practice to regard an auscultatory sign, which is not immediately recognized as a presystolic murmur, as an expression of one of the conditions now to be listed. The resemblance is created by the separate or conjoined effects of splitting, increased intensity of the first sound, and a systolic murmur.

Splitting of the first sound—This auscultatory sign, which is common in health or in the patient with a prolonged P-R or P-S period in the electrocardiogram, is often mistaken for a presystolic murmur. The likeness to mitral stenosis is increased when the splitting is accompanied by a double impulse in the upright posture which resembles a thrill

Hypertension.—Mitral stenosis and hypertension may be present in the same patient, but more often it means that when this dual diagnosis has been made, the altered first sound of hypertension has been mistaken for the presystolic murmur of mitral stenosis

Aortic incompetence.—Here a similar change in the first sound with the addition of a systolic murmur creates an auscultatory sign which closely simulates a presystolic murmur Naturally, should the aortic lesion be rheumatic, such a sign would suggest a diagnosis of mitral stenosis If it is non-rheumatic some would allude to it as an Austin Flint murmur, but a phonocardiographic study of this murmur has not shown it to be in any way distinctive (Fig 38)

Tachycardia.—The first sound in the mitral area is often loud and rough in tachycardia, especially that which arises in thyroid toxaemia, so that the presystolic murmur of mitral stenosis is simulated. When the heart rate slows, the auscultatory sign disappears



FIG 38.—Aortic incompetence Although a sound resembling a presystolic murmur (Austin Flint) was heard in this patient with luetic aortic incompetence, a murmur is not shown in the tracing in relation to the first heart sound. An early diastolic murmur (M) is recorded

SYSTOLIC MURMUR IN THE PULMONARY AREA

Pulmonary stenosis

This is a rough and loud murmur, moderately short in duration, and accompanied by a thrill. In the presence of a defect of the ventricular septum, forming part of Fallot's syndrome, it may be heard in the aortic area and even along the vessels of the neck, and the thrill may be wanting.

Patent ductus arteriosus

Like the murmur of pulmonary stenosis, this is rough in character and is accompanied by a thrill. It is a long murmur lasting through systole into diastole,

with accentuation of the second sound when the murmur is intensified. Such a murmur is pathognomonic of patent ductus arteriosus, and its continuous character has earned for it the descriptive terms of humming top, rolling sea, and machinery murmur.

Haemic murmur

The haemic murmur in the pulmonary area may be described in the same terms as the one appearing in the mitral area. It may only be present in the pulmonary area, but if it is also heard in the mitral area it is often as loud or louder.

Conducted murmurs

Aortic and mitral systolic murmurs are often audible in the pulmonary area, although not so loud, and it may be held that if a loud or harsh systolic murmur is of maximal intensity in the pulmonary area it signifies a congenital deformity connected with the pulmonary cusps or trunk. A systolic murmur in this area is often found in patients exhibiting a moderately severe defect of the auricular septum.

Innocent murmur

Apart from the murmur in this area which is associated with the innocent mitral murmur of reclining posture, a lone innocent pulmonary systolic murmur is often heard in young subjects. It is roughish or blowing in character and is louder in the reclining than upright posture. It may disappear to auscultation on deep breathing and it is never accompanied by a thrill.

DIASTOLIC MURMUR IN THE PULMONARY AREA

Aortic incompetence is the usual cause of a diastolic murmur in the pulmonary area. Often the murmur of aortic incompetence is better heard in the pulmonary than in the aortic area, and this is probably explained by the fact that the aortic valve is situated to the left of the midline. Whether the murmur is louder in one area or another cannot decide the actual cause of the aortic incompetence. *A diastolic murmur in the pulmonary area in a patient with mitral stenosis can* sometimes mean pulmonary incompetence (Graham Steell murmur), although the usual interpretation of such a murmur is that aortic incompetence is also present. Sometimes a pulmonary diastolic murmur signifies pulmonary incompetence from dilatation of the base of the pulmonary artery as in *auricular septal defect*, in this event the murmur is hardly ever heard to the right of the midline as is the case with the murmur of aortic incompetence. The diastolic murmur in patent ductus arteriosus is part of the characteristic machinery murmur. Occasionally a short early diastolic murmur is heard in pulmonary stenosis, and in so-called dilatation of the pulmonary artery.

SYSTOLIC MURMUR IN THE AORTIC AREA

Aortic stenosis

It is customary to regard a rough aortic systolic murmur as evidence of aortic stenosis if it is accompanied by a thrill. It is likely, however, that a rough murmur, even without a thrill, stands for a certain amount of thickening of the aortic cusps.

Aortic valve sclerosis

A roughish aortic systolic murmur, which is never loud or accompanied by a thrill, and is sometimes only elicited on direct auscultation in the upright posture in the aortic area, but is well heard in the mitral area, probably indicates slight thickening of the base of the aortic cusps. Naturally, such a lesion is immaterial in prognosis.

Conducted murmurs

The systolic murmur of pulmonary stenosis is often audible in the aortic area and it may be loud especially when there is a defect of the ventricular septum. The murmur of patent ductus arteriosus is often heard in the aortic area but never as loud as in the pulmonary area. A slight systolic murmur from anaemia or cardiac displacement may also present in the aortic area, and the remarks on such a murmur in the mitral area apply equally here.

DIASTOLIC MURMUR IN THE AORTIC AREA

Aortic incompetence is the only cause of a diastolic murmur in the aortic area, apart from the rare event of rupture of the sinus of Valsalva into a heart cavity or the pulmonary artery. It is soft in character, but if there is much thickening, with rigidity of the cusps (stenosis), the murmur is rough. The second sound may be impure or even accentuated, but more often the sound is obscured by the murmur. It is conducted down and to the left as far as the mitral area. It is often better heard in the pulmonary area *If the murmur of aortic incompetence is ever in doubt, and this is often the case when the lesion is early, it should be sought by direct auscultation at the lower end of the sternum with the breathing halted at the end of expiration and the patient in the upright posture.*

SYSTOLIC MURMUR IN THE TRICUSPID AREA

Tricuspid stenosis

If the mitral systolic murmur of mitral stenosis becomes less well heard towards the midline, and a murmur gets more obvious as the tricuspid area is approached, tricuspid disease is probably present , a thrill sometimes accompanies it.

Coarctation of the aorta

A systolic murmur in coarctation is not confined to the tricuspid area, for it is heard along the course of the hypertrophied internal mammary arteries, and is placed in late systole.

CHAPTER 3

CARDIAC ENLARGEMENT

In that cardiac enlargement predisposes to cardiac dysfunction and failure, it behoves the clinician, when estimating prognosis in a patient with cardiovascular disease, to determine the size of the heart. Prognosis does not depend solely on the degree of cardiac enlargement, but when the cause of the enlargement is known, its extent may often guide judgement on the outcome of the illness. The statement made by the clinician to his patient that " the heart is slightly enlarged " is ill-considered if it is meant to convey that the heart is only under suspicion and that there is nothing of serious import Indeed, if enlargement of the heart is present it demands a definition as to its cause, its degree, and its influence in prognosis. The finding of cardiac enlargement, particularly the estimation of its extent, is often difficult from clinical examination alone. The position of the apex beat is the best clinical index, but cardioscopy must ultimately decide the extent and direction of the enlargement, for it is important to delineate the right and posterior borders of the heart as well as its left border.

When the apex beat has shifted outwards to the left it is due to cardiac enlargement or displacement, but if it has moved to the right it is always the result of cardiac displacement The apex beat is always displaced to the left when the left ventricle is enlarged, but it needs emphasis that enlargement of the right side of the heart also displaces the apex beat to the left The extent to which the apex beat moves outwards in left ventricular enlargement depends more on distension of the cavity and less on the degree of hypertrophy of its walls.

Since the location of the apex beat assumes such great importance in determining cardiac enlargement by clinical examination, it is opportune here to enumerate the conditions which displace it. Most of these will be described separately in the text so that they will only be listed now with brief annotations on their effects upon the apex beat They belong to two main groups, those resulting from cardiovascular disease, and those arising outside the cardiovascular system With the exception of congenital dextrocardia the apex beat in the first group moves because of cardiac enlargement, and in the second group as a result of cardiac displacement.

CARDIOVASCULAR DISEASE DISPLACING THE APEX BEAT

Congenital disease

Dextrocardia.—In congenital dextrocardia the apex beat occupies its normal position as regards the rib space but it is on the right side Naturally this condition is a trivial abnormality rather than a disease.

Stenosis (coarctation) of the aortic arch.—The apex beat in this instance is displaced outwards by the effects of hypertension. The shift is more conspicuous when aortic incompetence has supervened, and this complication is not uncommon.

Subaortic stenosis.—As in the acquired type of aortic stenosis, so in this type the apex beat does not as a rule show great displacement because, in spite of considerable hypertrophy of the wall of the left ventricle, the distension of the cavity is not prominent in the early stage.

Right-sided enlargement.—Conditions which produce much distension of the right auricle and ventricle, notably auricular septal defect, may cause prominent displacement of the apex beat to the left.

Acquired disease

Aortic incompetence.—This should be mentioned first because in all but the early cases it displaces the apex beat conspicuously to the left.

Hypertension.—Papilloedemic hypertension usually causes obvious displacement of the apex beat from the start, but in simple hypertension the shift of the apex beat is not noticeable for a time.

Aortic stenosis.—Although there may be much hypertrophy of the wall of the left ventricle in aortic stenosis, its cavity in the initial stages does not show much distension so that the apex beat is not obviously displaced. When failure is present the displacement is greater.

Cardiac aneurysm.—In an elderly patient, when aortic incompetence and hypertension have been excluded as causes of a displaced apex beat, cardiac aneurysm following infarction may be an explanation

Mitral stenosis.—The left ventricle is not distended in mitral stenosis Should the apex beat be displaced outwards it should compel a search for aortic incompetence or hypertension If neither condition is present, it is safe to infer that distension of the right heart and left auricle has caused the heart to move and to rotate outwards displacing the apex beat in the same direction.

Arteriovenous aneurysm.—The arteriovenous aneurysm which causes enlargement of the heart and displaces the apex beat outwards is usually the acquired type with a moderately large fistulous opening When this is closed the heart diminishes in size rapidly and the apex beat resumes its normal position.

In the absence of hypertension, cardiac enlargement can seldom be made out on clinical examination in patients with thyroid toxaemia, emphysema, anaemia, myxoedema, cardiac infarction, or bradycardia, so that the apex beat is only slightly displaced ; it is only on cardioscopy that an increase in the size of the heart can be made out in these conditions Enlargement of the heart does not occur in adherent pericardium unless valvular disease is also present producing cardiac hypertrophy independently. Familial cardiomegaly is a rare cause.

NON-CARDIOVASCULAR DISEASE DISPLACING THE APEX BEAT

Affection of the lung or pleura

Fibrosis of lung.—Displacement of the apex beat is greater when fibrosis affects the right, rather than the left lung, when it may shift so far to the right as to merit the designation " acquired dextrocardia ". Often its position is unaltered by moderate fibrosis of the left lung.

Collapse of the lung.—Carcinomatous obstruction of a bronchus is an important cause of collapse of the lung, effecting great displacement of the apex beat to the corresponding side. A foreign body or a plug of mucus lodged in the bronchus will produce the same result. A lesser degree of displacement is found in pneumonia in children, from partial collapse of the affected lung.

Pleural effusion.—As a rule, displacement of the apex beat is not prominent in pleural effusion unless it is large.

Pneumothorax.—Air in the pleural cavity, provided it exerts a positive pressure and provided pleural adhesions are not numerous, displaces the apex beat to the opposite side.

Scoliosis involving the upper and middle thoracic regions is a very common cause of a shifted apex beat, and a realization of this is much needed. Although the shift occasionally takes place to the right, the common one is to the left. In many cases the spinal deformity is not obvious on clinical examination of the back, and greater attention should be paid to the set of the shoulders. The importance of scoliosis is not confined to explaining a displaced apex beat, but it is applicable to the interpretation of findings at cardioscopy. The common features of the two types of scoliosis may now be summarized.

In the **common type of scoliosis** (Figs. 39 and 40) the apex beat is displaced to the left, the left shoulder is higher than the right and the same applies to the

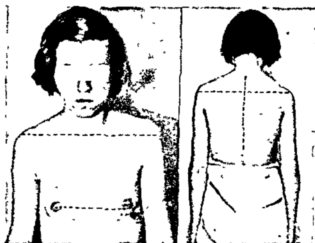
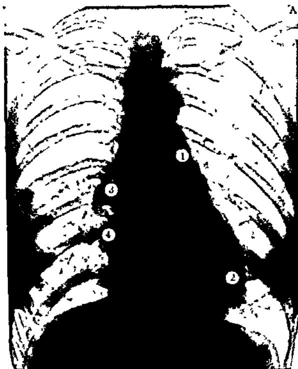


FIG 39.—The common type of scoliosis. The left shoulder and left nipple are higher than those of the right side, and the apex beat (marked with dot) is displaced to the left, and caused a false *diagnosis of cardiac enlargement in this child aged 12 years. The scoliosis is seen to better* advantage in the front view.

level of the nipples. Corrective manoeuvre at cardioscopy should turn the patient to the right.

In the **uncommon type of scoliosis** (Figs. 41 and 42) the apex beat is displaced to the right or sometimes to the left, the right shoulder is higher than the left,



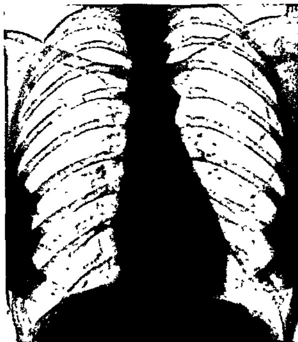


Fig 40—Usual type of scoliosis displacing the heart to the left (A), causing prominence of the pulmonary artery (1) and left ventricle (2), and exposing the root of the right lung (3) and the right border of the spine (4) Slight rotation of the subject to the right (B) has corrected such discrepancies Healthy female aged 23 years whose apex beat was displaced to the left

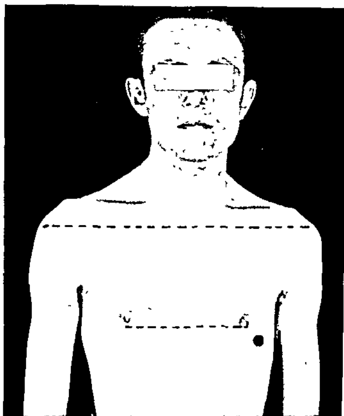


Fig 41 —The uncommon type of scoliosis The right shoulder and nipple are higher than those of the left side The apex beat in this kind may be displaced to the right or to the left (marked with a dot) as in this case

and the same applies to the level of the nipples. Corrective manoeuvre at cardioscopy should, as a rule, turn the patient to the left.

Often displacement of the apex beat to the left is due to thoracic asymmetry, when the rib spaces will appear narrower on radiological examination on one side of the chest than on the other

Depression of sternum —When 16 adults in whom there was moderate or greater degree of depression of the sternum were examined especially for its effect on the heart, it was found that the association of a displaced apex beat and a systolic murmur or splitting of the first heart sound with the deformity had led to a restriction of physical activities in every case. This invalidism had been enforced more rigidly whenever radiological examination had been added, because it had shown a big heart shadow. The symptoms were never once the result of heart enlargement or embarrassment, but were the direct outcome of the physical inactivity or mental anxiety resulting from unwarranted invalidism.

According to the extent of the depression the cases were separated into three groups (Fig 43). In *funnel depression* (Fig. 44), in which the hollowing was deep

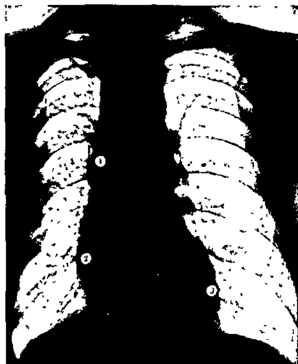
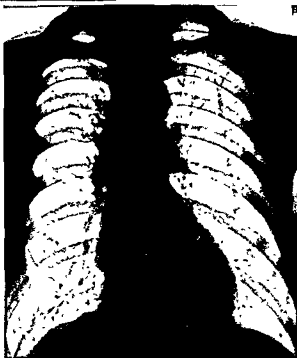


FIG 42—Unusual type of scoliosis displacing heart to right (A) and causing prominence of ascending aorta (1) and right auricle (2), the left ventricle (3) appears small Slight rotation of the patient to the left (B) corrects these discrepancies



the external antero-posterior chest measurement was from $4\frac{1}{2}$ to 5 inches In *cup depression* (Fig. 45), in which the recession was moderately deep and its apex rounded, the external antero-posterior measurement was from 5 to 6 inches. In *saucer depression* (Fig. 46), in which the dip was shallower and wider, the external antero-posterior measurement was from 6 to $6\frac{1}{4}$ inches. Depression without a reduction of the antero-posterior measurement to $6\frac{1}{4}$ inches or less has no effect upon the heart.

The radiological findings were characteristic for each group. Thus, in funnel depression the heart was displaced bodily to the left of the spine and was unchanged

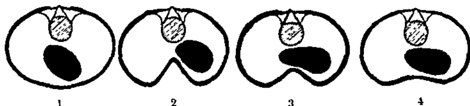


Fig 43 —Diagrammatic representation of the three kinds of sternal depression 1 Normal 2 Funnel depression. 3 Cup depression 4 Saucer depression.



Fig. 44 —Funnel depression of sternum Antero-posterior chest measurement of 5 inches against normal control of 8 inches



Fig 45 —Cup depression of sternum Antero-posterior chest measurement of $5\frac{3}{4}$ inches against normal control of 8 inches

in size or in shape (Fig. 47) In the cup depression the cardiac silhouette in the anterior view was less dense than normal and was moderately enlarged with prominence of the pulmonary arc ; in the left oblique view the heart shadow was normal or small, and was displaced backwards to overlap the spine, thereby



Fig 46—Saucer depression of sternum. Antero-posterior chest measurement of 6 inches against a normal control of 7½' inches.

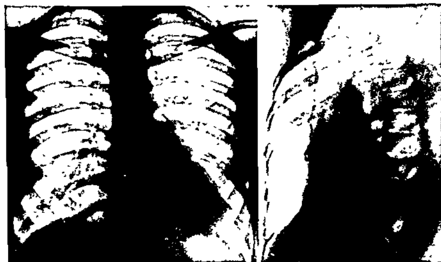


Fig 47.—Funnel depression of the sternum displacing the heart bodily to the left in the anterior view, and backwards to overlap the spine in the left oblique view.



Fig. 48.—Cup depression of the sternum pancaking the heart in the anterior view, and displacing it backwards to overlap the spine in the left oblique view

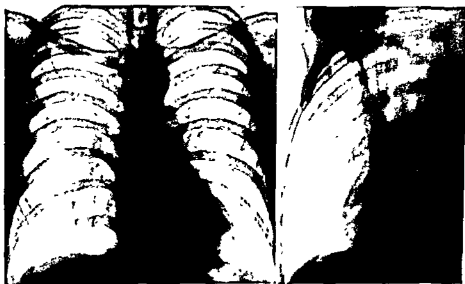


Fig. 49.—Saucer depression of the sternum causing some enlargement of the heart shadow in the anterior view, displacing it backwards to overlap the spine in the left oblique view



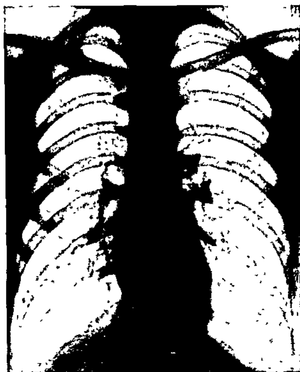


FIG 50—Alteration of the heart's contour in relation to the shape of the chest In (A) the heart is squat and the apex beat displaced to the left because of the wide and shallow chest In (B) the heart hangs in the middle of a narrow and long chest.

impoverishing the picture by obliterating the clear areas provided by the aortic window and the retrocardiac space (Fig. 48) Similar effects, although less in degree, were found in the saucer depression (Fig. 49).

It needs to be emphasized that none of the patients with depression of the sternum showed actual enlargement of the heart or suffered from symptoms relating to the heart. Indeed, their only handicap lay in the restriction imposed on them by a medical examination that had misinterpreted the clinical and radiological signs.

Raised diaphragm —With a wide and shallow chest the heart is pushed upwards and the apex beat displaced outwards (Fig. 50) Distension of the abdomen raises the diaphragm and displaces the heart upwards and slightly to the left Common conditions elevating the diaphragm in this way are abdominal obesity, ascites, distended liver from any cause, pregnancy, abdominal cysts and massive tumours. A distended stomach raises the left dome of the diaphragm

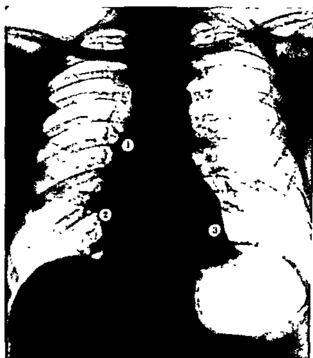


Fig. 51 —Gaseous distension of stomach raising dome of left diaphragm and displacing heart to right This effect causes prominence of ascending aorta (1) and right auricle (2), and makes the left ventricle (3) appear small.

so that the heart is displaced upwards and to the right (Fig. 51). Eventration of the diaphragm (congenital elevation of one lobe, usually the left) will do the same thing.

Diaphragmatic hernia is a rare cause of apex beat displacement, although the shift when it occurs may be a prominent one. The apex beat moves according to the dome through which the intestine herniates, usually the left, and the certain diagnosis can only be established on radioscopy (Fig. 52).

Other causes

A cyst or teratomatous tumour of the lung, and lymphogranuloma of the mediastinum, may be so massive as to produce the same effect through the medium of pleural effusion. A large aneurysm of the descending aorta will displace the heart forwards and slightly to the left when the shifted apex beat

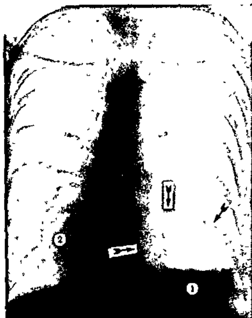


FIG 52.—Herniation of the stomach (outlined by arrows), containing barium (1) in its lower part, through the left dome of the diaphragm, displacing the heart to the right. The right auricle (2) is made more prominent

also becomes diffuse and thrusting. The apex beat is often difficult to locate in a patient with emphysema, but sometimes in those where it can be felt it may be found internal to its normal position and slightly lower on account of the depressed diaphragm.

Dilatation of a heart chamber generally takes place in association with hypertrophy of its walls. It is a gradual process and in no sense is it a sudden expansion from "myocardial weakness." Common examples of cardiac dilatation are provided by distension of the left ventricle in aortic incompetence and in hypertension, especially when heart failure is present, left auricle in mitral stenosis, and right auricle and ventricle in auricular septal defect. In all these conditions the process of cardiac enlargement or distension takes place gradually. It is in children that the terminology of "acute cardiac dilatation" is sometimes applied, especially in acute nephritis and certain of the specific fevers, in these circumstances it is necessary to consider the influence of tachycardia and the effects of heart failure upon the position of the apex beat before nominating dilatation as the cause of its displacement.

CHAPTER 4

ARRHYTHMIA

WHEN the heart beat and the pulse are constant in force, and bear an unvarying relationship to one another at 60 to 100 per minute, and when the electrocardiogram shows no dissociation between auricular and ventricular systole, the heart action conforms to the normal or physiological. Any departure from this standard of normality constitutes arrhythmia. The peculiar features of this important group of cardiac disorders are best considered under four main headings, which are summarized here with the addition of sub-headings.

Altered vagal or sympathetic influence

Sinus bradycardia
Sinus tachycardia
Sinus arrhythmia
Sino-auricular block

Shifting of focus normally producing cardiac impulse

Extrasystoles
Auriculo-ventricular nodal rhythm
Reciprocal rhythm
Paroxysmal tachycardia (auricular tachycardia) and auricular flutter
Auricular fibrillation
Ventricular fibrillation

Faulty conduction of cardiac impulse

Delayed auriculo-ventricular conduction (long P–R period)
Accelerated auriculo-ventricular conduction (short P–R period)
Incomplete auriculo-ventricular block
Complete auriculo-ventricular block
Bundle branch block

Faulty heart contraction (pulsus alternans)

ALTERED VAGAL OR SYMPATHETIC INFLUENCE

The actual part played in health and disease by the sympathetic nerve fibres and the intrinsic cardiac ganglia is not known. Our knowledge of vagal control, although limited, is more definite. The vagus acts as an inhibitory nerve and it normally restrains the stimulus-producing centre (pacemaker of the heart) situated in the sino-auricular node. Stimulation of the vagus by pressure on the carotid sinus in the neck slows the heart rate, whereas depression of the vagus, by atropine for instance, quickens it. Four types of altered heart action belong to this group.

SINUS BRADYCARDIA

When the rate is 60 or less per minute and the cardiac impulse arises in the sino-auricular node, the rhythm is designated sinus bradycardia. The condition probably arises from increased vagal tonicity and is found in healthy subjects, especially young athletes, and in patients with increased intracranial pressure, certain specific fevers, notably typhoid, and sometimes jaundice. Vagal irritation from tumours in the neck or mediastinum may produce it.

Sinus bradycardia assumes importance in directing attention to the disease causing the slow rate, or when it is to be distinguished from the more serious

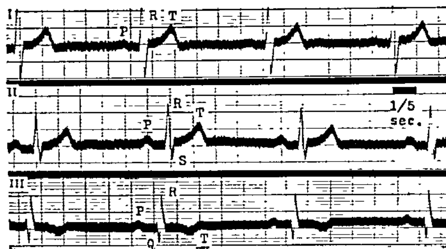


FIG 53.—Sinus bradycardia (about 50 a minute) from a healthy male aged 20

form of bradycardia due to heart block If the rate is over 50 per minute, sinus bradycardia is the likely explanation, but if under 40, heart block is the usual cause. Exercise or the administration of drugs which habitually quicken the heart rate will more readily induce tachycardia in a patient with sinus bradycardia than in one with heart block

The *electrocardiogram* (Fig. 53) is distinctive and demonstrates the sinus origin of the impulse as well as the slow rate. Rarely, the rate in sinus bradycardia may be so slow as to allow the idioventricular pacemaker to initiate an impulse producing a ventricular systole ; this is known as ventricular escape (Fig 54)

Cardioscopy shows a full-sized heart (Fig. 55) and its outline may be stencilled It is unwise to refer to this as enlargement for such a term should be reserved for the pathological variety. It is only necessary to be aware that a relatively big cardiac silhouette is expected at cardioscopy in a case showing bradycardia. The condition itself calls for no treatment

SINUS TACHYCARDIA

When the rate is 100 or more per minute and the cardiac impulse arises in the sino-auricular node, the rhythm is designated sinus tachycardia. It is found in

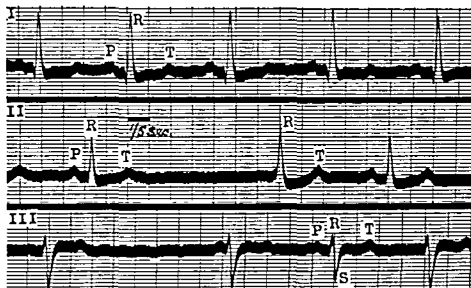


FIG 54 —Ventricular escape The second beat in lead II is delayed and it shows no auricular wave The cardiogram also shows sinus bradycardia (about 64 a minute)

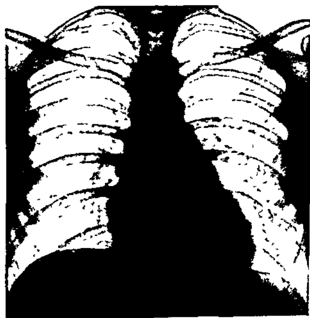


FIG 55.—Sinus bradycardia (65 beats a minute) in a healthy youth aged 18 years There is slight enlargement of the cardiac shadow

health, especially in young or nervous subjects, and in thyroid toxaemia, illnesses with pyrexia, chronic infection, particularly pulmonary tuberculosis, and in debilitating conditions. Drugs, notably atropine, adrenaline, ephedrine, trinitrin, and thyroid, readily produce sinus tachycardia.

Like sinus bradycardia, the condition assumes importance when it directs attention to the disease causing it, or when it is necessary to differentiate it from the other form of regular rapid heart action, namely, paroxysmal tachycardia. If the rate is 160 or more per minute paroxysmal tachycardia is the likely explanation, and if less than 150, sinus tachycardia may be the explanation, although an

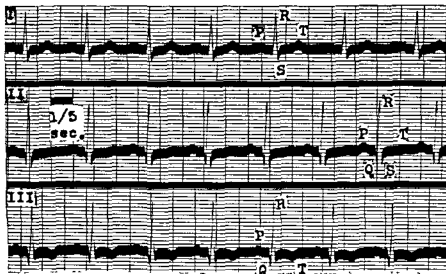


FIG 56—Sinus tachycardia (about 115 a minute) The T wave is low in lead II and inverted in lead III. The tracing is from a healthy subject

electrocardiogram is needed to exclude auricular tachycardia with a relatively slow ventricular rate Like bradycardia, agents which accelerate the heart will help to tell whether the tachycardia is of the sinus or of the auricular kind. Thus, in sinus tachycardia, exercise will raise the rate still higher and rest will slow it, such influences are without much effect in auricular tachycardia

The *electrocardiogram* (Fig 56) is distinctive and demonstrates the sinus origin of the cardiac impulse as well as the rapid rate. At a high rate it is advisable to include the CR_1 lead for the purpose of differential diagnosis (Fig 57). *Cardioscopy* shows no cardiac enlargement unless there is associated heart disease.

Treatment should be directed to the cause. If it is due to thyroid toxaemia, subtotal thyroidectomy will remove it. In the case of nervous subjects, when palpitation is a common symptom, it may be allayed in great measure by the use of a sedative drug such as phenobarbitone

SINUS ARRHYTHMIA

When a variation in the heart rate takes place during inspiration and expiration the condition is known as sinus arrhythmia. The pulse accelerates during inspira-

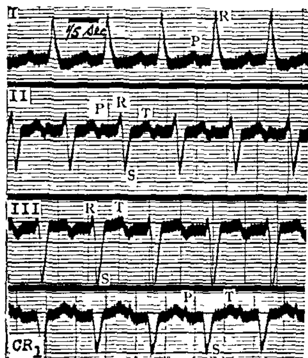


FIG 57 —Sinus tachycardia at a high rate (165) The CR_1 cardiogram confirms the sinus origin of the impulse

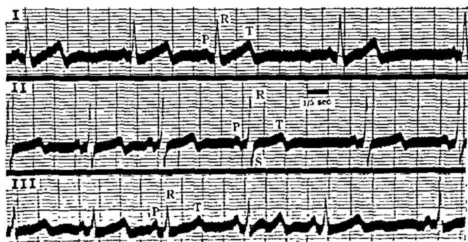


FIG. 58 —Sinus arrhythmia In each lead the rate quickened to 85 a minute during inspiration and slowed to 58 during expiration ; from a healthy child aged 9 years.

tion and slows with expiration. It is likely that the alternate quickening and slowing is caused by altered tonicity of the vagus, so that during inspiration vagal tonicity is lessened, resulting in tachycardia, and during expiration the tonicity returns to normal when the tachycardia subsides. Although the condition is met with in heart disease, it is a common finding in health, and it is to be regarded as a natural or physiological variation. It assumes greatest importance in diagnosis from heart block itself and the problem usually presents in the case of children, in whom sinus arrhythmia is best exhibited. Tachycardia induced by exercise will usually correct the abnormal rhythm The *electrocardiogram* (Fig. 58) will establish the diagnosis by showing the absence of block.

SINO-AURICULAR BLOCK

Sino-auricular block is an example of dropped or missed beat, so that the interval between two auricular systoles is approximately equal to two normal cardiac

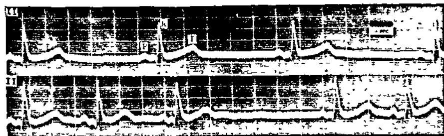


FIG 59 —Sino-auricular block. In the first curve, alternate beats are missing, but there is only one missed beat in the second tracing.

cycles. This intermission may happen frequently or only occasionally. The condition is probably caused by depression of the sino-auricular node consequent on increased vagal tonicity. The recognition of the altered rhythm is only possible by *electrocardiography* (Fig. 59) and the prognosis associated with it depends entirely on the clinical condition, for example, rheumatic fever, which has induced it. It often appears in healthy subjects.

SHIFTING OF FOCUS NORMALLY PRODUCING CARDIAC IMPULSE

EXTRASYSTOLES

The term, extrasystole, introduces a certain ambiguity in that the prefix extra does not mean an added beat, but a beat arising in a focus outside the usual seat of impulse-formation in the sino-auricular node—an ectopic beat. As a rule *an extrasystole arises prematurely and replaces the succeeding normal systole* so that the abnormal beat is followed by a compensatory pause. Rarely, an extrasystole may take place within the normal diastolic phase between two normal systoles and it is then known as an interpolated beat

Aetiology

In the greater proportion of cases a cause cannot be found either in the heart or elsewhere.

Direct stimulation of the heart by *injury, or experimentally by means of an* electrified terminal, will initiate extrasystoles. This method has been applied to the human heart, exposed during drainage of suppurative pericarditis, for the purpose of studying the electrocardiogram obtained when stimulating different areas of the heart. Extrasystoles may happen during abdominal operations or may follow an exaggerated excursion of diaphragm during deep inspiration.

Fatigue probably plays a part in increasing the incidence of extrasystoles in a subject already exhibiting this arrhythmia.

Excessive smoking, and digitalis, may induce extrasystoles or increase their frequency when they are already a feature. They sometimes appear in acute rheumatism or diphtheria and during chloroform anaesthesia.

Sudden undue exertion in one unaccustomed to physical exercise will often give rise to extrasystoles

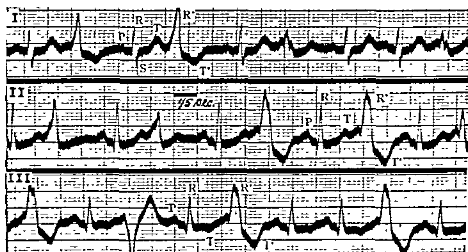


Fig 60 —Frequent extrasystoles in the presence of sinus tachycardia from an otherwise healthy subject

Symptoms and diagnosis

There may not be any symptoms and the patient may remain unaware of the arrhythmia even though examination shows frequent extrasystoles When a patient is conscious of the abnormal rhythm, the palpitation experienced is variably described as " missing, thumping, or stopping of the heart," and these sensations usually become more noticeable when in bed at night. In a third group the extrasystoles are *frequent and the patient is apprehensive so that more severe symptoms,* such as faintness and distress, make their appearance.

Extrasystoles are easily recognized when appearing frequently Often they fail to produce a pulse at the wrist although audible on auscultation of the heart. Over shorter or longer periods they may recur at regular intervals and sometimes will alternate with normal systoles producing a coupling rhythm (pulsus bigeminus) In this circumstance, if the pulse produced by the extrasystole is smaller than

the one from the preceding normal systole, the condition of pulsus alternans may be simulated; differentiation rests with the discovery of a longer pause after the smaller beat (compensatory pause following a premature beat), but if the pause is not obvious resort should be made to electrocardiography. Extrasystoles may appear at irregular intervals, and in this event, if they recur frequently, auricular fibrillation is simulated In fact, the telling of frequent extrasystoles from auricular fibrillation forms an important clinical problem and the following desiderata will prove valuable in the diagnosis:

1. If the altered rhythm is caused by multiple extrasystoles, palpitation is likely to be the outstanding symptom, and if due to rapid auricular fibrillation the symptoms of heart failure are likely to be present.

2 If the arrhythmia appears in a patient with mitral stenosis, hypertension or thyroid toxaemia, auricular fibrillation is usually the explanation

3 When tachycardia induced by exercise or other means annuls the abnormal rhythm, the irregularity has been caused by extrasystoles Occasionally, however, extrasystoles may appear in the presence of moderate tachycardia (Fig 60)

4 If the irregularity takes place in brief paroxysms extrasystoles will prove to be the usual cause

5 Electrocardiography will decide the nature of the arrhythmia which may often evade clinical diagnosis till this test is carried out.

Electrocardiographic types

The ectopic stimulus initiating a premature systole may arise in the auricle, in the

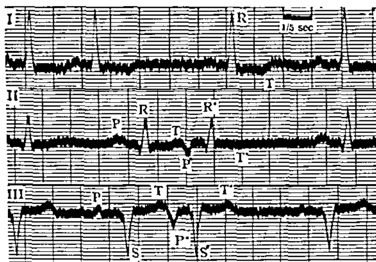


FIG 61.—Auricular extrasystoles. In the premature beats in leads II and III the P wave is inverted and the P–R period is shortened

auriculo-ventricular node, or in the ventricle, and accordingly three distinctive electrocardiograms are obtained.

Auricular extrasystole.—In this type the auricular wave (P) in the electrocardiogram (Figs 61 and 62) is written prematurely, and since it is produced by a stimulus arising in a part of the auricle removed from the sino-auricular node, the wave is deformed and sometimes inverted

The P-R period is usually slightly altered and the ventricular complex shows a slight variation from the normal one for that patient. Rarely, the auricular beat is so premature that the impulse arrives in the ventricle before this has completed its systole following the normal sinus impulse so that the ventricle does not respond to the premature auricular beat (blocked auricular premature beat) (Fig 63)

Auriculo-ventricular nodal extrasystole.—In this type the P wave in the electrocardiogram (Fig 64) is altered and is placed either before or after, or may coincide with, the QRS of the *ventricular complex*

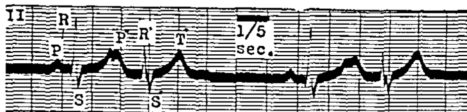


Fig. 62 —Auricular extrasystoles The P wave in the premature beats is upright and the P-R period is prolonged



Fig 63 —Blocked auricular extrasystoles In two beats a premature P wave is written at the commencement of the T wave, but its corresponding ventricular waves fail to appear

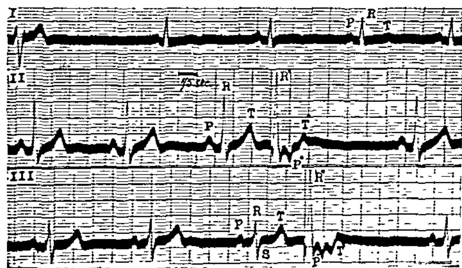


Fig. 64 —Nodal extrasystoles The P wave, which is inverted, follows the QRS complex.

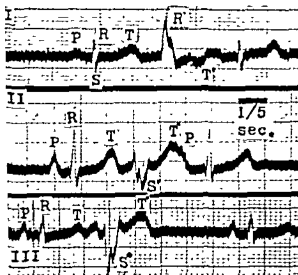


Fig 65—Right ventricular extrasystoles Aberrant QRS complexes of premature beats are directed upwards in lead I and downwards in lead III

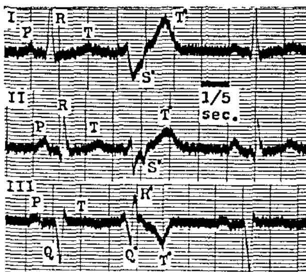


Fig 66—Left ventricular extrasystoles Aberrant QRS complexes of premature beats are directed downwards in lead I and mainly upwards in lead III

Ventricular extrasystole.—A ventricular extrasystole is denoted in the electrocardiogram by a wide QRS complex, not preceded by a P wave, and followed by a conspicuous T wave (known as a secondary T wave) written in the opposite direction to the QRS deflection. If the impulse producing the abnormal systole arises in the right ventricle, the abnormal QRS complex is upright in lead I and down in lead III (Fig. 65) If the impulse arises in the left ventricle the QRS is down in lead I and upright in lead III (Fig. 66).

When the electrocardiogram in cardiac infarction is equivocal as, for instance, in bundle branch block, the form of an extrasystole may provide a clue to such a diagnosis (Fig. 67)

Treatment

If the arrhythmia comes to the notice of a clinician during routine physical examination, in a patient without palpitation, no reference should be made to it.

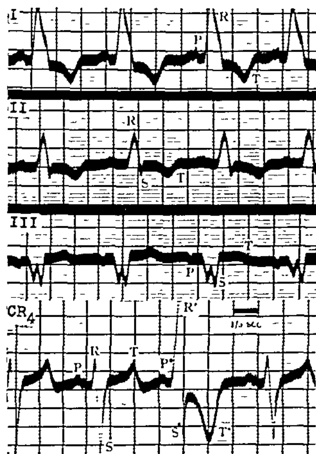


Fig. 67.—Left bundle branch block from cardiac infarction. The form of the T wave of the extrasystole in CR_4 provides the information that the bundle lesion has resulted from cardiac infarction

Patients who are aware of the irregular heart action are classified into three groups for the purpose of treatment. To the first group belong those who react favourably

to reassurance on the innocent nature of the condition. In the next group are the nervous patients requiring a sedative such as half a grain of phenobarbitone twice daily for a time, in addition to reassurance In the third group the extrasystoles are so frequent and persistent as to cause real distress, calling for an attempt to correct the abnormal rhythm In these cases due regard must be paid to excessive smoking and recent heavy exertion as causes before resorting to quinidine treatment which sometimes yields satisfactory results.

AURICULO-VENTRICULAR NODAL RHYTHM

Persistent auriculo-ventricular nodal rhythm is uncommon and its recognition is only determined with certainty by electrocardiography. Temporary shifting of the pacemaker to the node is a common event when it sometimes produces occasional coupling of the pulse. It may happen in the absence of heart disease and it is of no consequence in prognosis.

The auricular wave in the *electrocardiogram* (Fig. 68) is often inverted and as

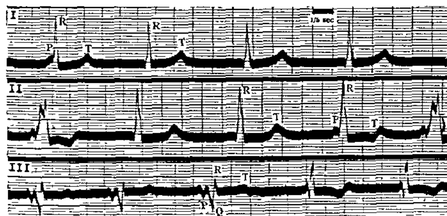


Fig 68 —Nodal rhythm with wandering of the pace-maker The P wave is variably written before, at the beginning of, or within the QRS complex Deformity of the T wave is greatest following the widest QRS complexes

a rule it follows the QRS complex, although it may coincide with or precede it. The P–R or R–P periods vary in duration in the same tracing and so does the P–P period, but the R–R period may remain constant. The variable position of the P wave is probably due to a variation in the retrograde conduction of the impulse into the auricle and not to a shifting of the seat of origin of the impulse in the node.

RECIPROCAL RHYTHM

In this arrhythmia a single impulse arising in the auriculo-ventricular node gives rise to two ventricular contractions. The first results from the impulse passing forward to the ventricle from the node in the normal way. The backward transmission to the auricle is so slow (retrograde heart block) that after the auricular contraction the impulse is able to pass down to reach the ventricle after recovering from its refractory phase and it contracts again

In itself this form of abnormal rhythm has no special significance and the prognosis in a case presenting it should be based on the underlying heart condition.

PAROXYSMAL TACHYCARDIA (AURICULAR TACHYCARDIA) AND AURICULAR FLUTTER

Since the right pectoral lead (CR_1) in patients with paroxysmal tachycardia has shown the common incidence of A–V dissociation, the terms paroxysmal tachycardia and auricular flutter have almost become interchangeable.

Thus, *auricular flutter* is paroxysmal tachycardia in which a moderate auricular rate (200–260) facilitates the finding of A–V dissociation in the cardiogram because both auricular waves are separate from the two main waves of the ventricular complex, a greater A–V dissociation from slowing of the ventricular rate shows three or more consecutive auricular waves.

Again, *paroxysmal tachycardia* is auricular flutter in which the more rapid auricular rate (260–500) prejudices the recognition of the auricular waves hidden within the ventricular complexes and hinders the discovery of a 2 to 1 A–V dissociation. In such circumstance comparison of a right pectoral cardiogram in an attack with one in normal rhythm will help in deciphering the nature of the arrhythmia.

From the clinical standpoint the attacks in the first group are inclined to be longer, more amenable to digitalis influence, and more certain to be associated with heart disease, but to each of these three features there are frequent exceptions, and the common unity of paroxysmal tachycardia and auricular flutter appears to be established with auricular tachycardia as the essential mechanism of each.

Aetiology

As in the case of extrasystoles, it is often impossible to find the cause of paroxysmal tachycardia, but unlike extrasystoles this altered rhythm is more frequently identified with heart disease, particularly mitral stenosis, hypertension, cardiac infarction and thyroid toxaemia. Excessive smoking, indigestion, fatigue or undue exertion, may sometimes precipitate attacks. In a small number of patients the electrocardiogram shows characteristic changes in the form of a prolonged QRS complex similar to that found in bundle branch block, and a short P–R period.

Symptoms and diagnosis

Palpitation is the outstanding symptom of paroxysmal tachycardia and is usually described as taking place in attacks These may be brief and last only a few minutes, or they may continue for hours, days, or even weeks. Rarely, the paroxysm lasts for many months. A characteristic feature of the attack is the sudden onset and usually the abrupt ending ; exertion often precipitates the paroxysm. In addition to palpitation, faintness, weakness and distress are common symptoms, and few are able to follow their customary occupation during the attack. If vomiting takes place it may abort the attack. Occasionally the paroxysm terminates with cardiac standstill before normal rhythm is restored, so that the patient may become unconscious for a brief period (Stokes-Adams attack) Sometimes the characteristic pain of cardiac ischaemia is experienced and is relieved as soon as the paroxysm abates ; this symptom of pain in paroxysmal tachycardia does not indicate coronary disease, nor does it predict for the case



FIG 69—Paroxysmal tachycardia Enlargement of the heart and failure (A) after an attack of tachycardia lasting a week It resumed its normal size (B) after a week in normal rhythm



which exhibits it a poorer prognosis. In the presence of a cardiac lesion persistence of the arrhythmia expedites heart failure, but even without heart disease failure may set in when the attack has lasted many days (*Fig. 69*).

Sinus tachycardia may sometimes be difficult to diagnose from paroxysmal tachycardia, but attention to the following clinical details should make this possible in many instances without the aid of electrocardiography.

1. The heart rate rarely exceeds 160 a minute in sinus tachycardia and is usually much less, whereas in paroxysmal tachycardia it is usually much greater, although it may sometimes be less.

2 The pulse in sinus tachycardia accelerates gradually and in response to such stimuli as exercise and emotional disturbance, whereas in paroxysmal tachycardia it quickens abruptly and is not much accelerated by exertion

3 The quickened pulse in sinus tachycardia subsides gradually, especially when the patient is rested, whereas the attack of paroxysmal tachycardia ends abruptly, and rest has no influence on the rate

4 Maintenance of a uniformly high heart rate is a characteristic feature of paroxysmal tachycardia but it is not found in sinus tachycardia

5 In some cases in which the ventricular rate is less than 100 a minute it might be possible to tell the much more rapid action of the auricles from the venous pulse in the neck

6 In thyroid toxaemia, although sinus tachycardia is usual, paroxysmal tachycardia may also supervene, when iodine administration is more likely to affect the rate in the sinus than it is in the paroxysmal type

7 The presence of heart disease, especially mitral stenosis, would as a rule favour a diagnosis of paroxysmal tachycardia

The auricular rate in paroxysmal or auricular tachycardia varies from 250 to 500 per minute, but when the ventricular rate is also rapid, all the auricular contractions are difficult to identify ; the recognition of the auricular beats, however, is facilitated by recording the right pectoral chest lead (CR_1) during tachycardia and comparing it with the record obtained in normal rhythm. When this test becomes routine a high incidence of A–V dissociation will be found among the cases. A regard of the auricular rate and the degree of A–V dissociation, both of which determine the conspicuity of the auricular waves in the tracing, places the electrocardiograms of auricular tachycardia in five groups. In the past it has been traditional to allude to the first two groups as auricular flutter, but the mechanism appears to be the same for these as for the next two groups

1. **Auricular tachycardia of high rate with 3 to 1 (or greater) A–V dissociation.**—In this group the auricular rate is rapid (up to 300 per minute), but the ventricular rate is relatively slow (under 100), so that A–V dissociation is 3 to 1 or greater and three or more consecutive auricular waves are written outside the ventricular waves (Figs 70 and 71)

2 **Auricular tachycardia of high rate with 2 to 1 A–V dissociation.**—In this group the auricular rate varies from 200 to 260, and the ventricular rate from 100 to 130, both auricular waves are seen outside the ventricular waves (Figs 72 and 73)

3 **Auricular tachycardia of higher rate with 2 to 1 A–V dissociation.**—In this group the auricular rate varies from 260 to 400, and the ventricular rate from 130 to 200, alternate auricular waves fall within the S or T waves of the ventricular complex (Fig. 74)

4 **Auricular tachycardia of very high rate with 2 to 1 A–V dissociation.**—In this group the auricular rate is over 400 and the ventricular rate over 200, both auricular waves fall within the S and T waves of the ventricular complex (Fig 75)

5. **Auricular tachycardia of high rate without A–V dissociation (Parasinus tachycardia).**—In this group the auricular rate is rapid and the ventricular rate is the same so that the impulse from the ectopic focus travels through the sino-auricular node Such paroxysms are brief as a rule and recur frequently (Fig 76)

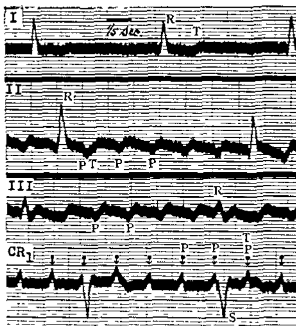


FIG 70—Auricular tachycardia of high rate with slow ventricular rate leading to 6 to 1 A-V dissociation

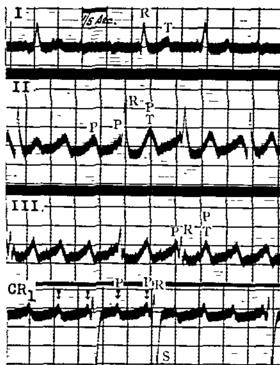


FIG 71.—Auricular tachycardia of high rate with moderate ventricular rate leading to 2 to 1 to 4 to 1 dissociation

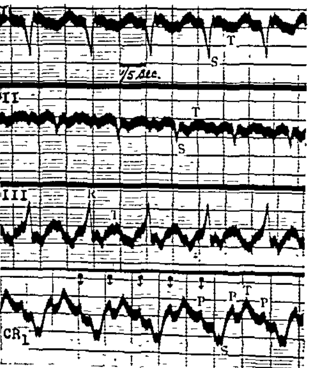


FIG 72.—Auricular tachycardia of high rate with 2 to 1 A-V dissociation which is best seen in lead CR_1.

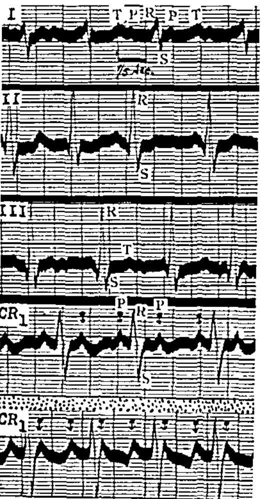


FIG 73.—Auricular tachycardia of high rate with 2 to 1 A-V dissociation which is best seen in lead CR_1; two such leads are shown, illustrating how a slower ventricular rate permits a better view of both auricular waves

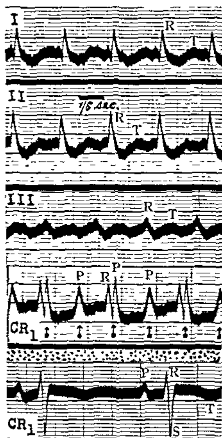


FIG 74 —Auricular tachycardia of higher rate with 2 to 1 A–V dissociation which is best seen in lead CR_1 which is compared with the same lead in normal rhythm

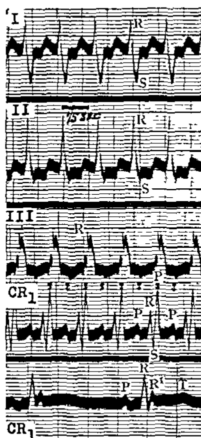


FIG 75 —Auricular tachycardia of very high rate with 2 to 1 A–V dissociation which is best seen in lead CR_1 which is compared with the same lead in normal rhythm

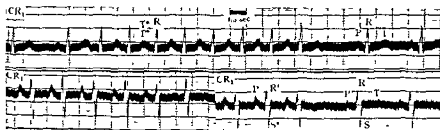


FIG 76 —Parasinus tachycardia Auricular tachycardia of high rate without A–V dissociation The paroxysms recur and are short lived

Prognosis

The outlook in any case of paroxysmal tachycardia depends largely on the presence or absence of heart disease as determined clinically and graphically. Thus, when heart disease is absent there is, as a rule, no cardiac enlargement or failure and the prognosis is good. A few cases, however, show heart failure when the attack has lasted many days, but in these instances the failure recedes as soon as the attack subsides When heart disease is present, the attack will soon precipitate failure, and the outlook then depends on the severity of the underlying heart disease and on the duration of the tachycardia.

Treatment

If an obvious cause of paroxysmal tachycardia is operating, such as excessive smoking, undue exertion, or thyroid toxaemia, correction of this must be a first consideration in treatment When the arrhythmia has supervened on heart disease, precipitating heart failure, it will demand its own specific therapy. In the absence of heart disease, treatment needs to be directed to the attacks. Here, as in the case of extrasystoles, reassurance will play an important part in allaying the patient's natural concern about the heart, and often no other treatment will be necessary if the paroxysms are brief. Next, an attempt may be made to stop the actual attack, but although several methods are recommended none has given consistently satisfactory results. Deep breathing or bending, pressure on either carotid sinus in the neck, pressure on both eyeballs, and the administration of a strong carminative mixture, are methods which have been tried and sometimes succeed. Ipecacuanha, by inducing vomiting, and acetylcholine derivatives, have often brought attacks to a close, but if they last, or even when brief and recurring frequently, quinidine or digitalis therapy holds out the best prospect of changing the abnormal rhythm. Even in the presence of heart failure as in hypertension, quinidine will often bring an attack to a close, so that in this condition at least, heart failure is not to be held as contra-indicating its use. Quinidine taken regularly with the object of preventing attacks is seldom successful, although it may be tried. In auricular tachycardia which lasts for some time, digitalis may often convert it into auricular fibrillation, when it may remain in this rhythm with advantage or revert to normal rhythm. If the attack is long, or if it is associated with failure, the patient should be in bed during the period when treatment is first started, and the services of an electrocardiograph should be available. Concerning the continuous medical treatment of the lengthy attack of auricular tachycardia, the following summary will be a guide to the management of most cases, although it will need to be modified in individual patients. When paroxysmal tachycardia is not accompanied by heart failure and quinidine has restored normal rhythm, the drug may be withdrawn after two months. When the attack fails to respond to quinidine, digitalis should be tried. If quinidine has converted auricular tachycardia in the presence of heart failure into normal rhythm, digitalis and mercurial diuretics should then be given for the relief of the failure signs. If the tachycardia in the presence of failure has changed into auricular fibrillation during digitalis therapy, digitalisation should be maintained irrespective of whether the rhythm continues as fibrillation or has reverted into normal rhythm.

AURICULAR FIBRILLATION

In auricular fibrillation the wave of contraction circulates continuously through the auricle constituting circus movement (Fig 77), but unlike auricular tachycardia it travels over a variable path and at a faster rate. The right pectoral chest lead (CR_1) permits the limited definition of auricular fibrillation as a succession of

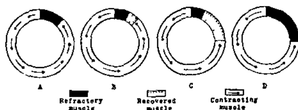


FIG 77.—A scheme to explain the mechanism of circus movement (A) Normal auricular contraction, when the excitation wave has completed a cycle it ends when it meets refractory muscle (B) Circus movement; the excitation wave at the end of a cycle meets recovered muscle so that it continues to travel round and round the auricle (C) Conduction rate slowed, this effect, produced by digitalis and quinidine, tends to establish circus movement (D) Refractory period lengthened; this effect produced by quinidine tends to terminate circus movement

auricular waves at a rate of about 400 per minute, differing slightly in size and shape and with a rhythm almost as often regular as irregular. The ventricular responses in fibrillation have no constant relationship to the auricular waves, as in auricular tachycardia, probably because the auricular rate is too high for any regular control

Like auricular tachycardia, the condition may occur in paroxysms or become established, and the paroxysmal type does not always emerge into the established type.

Aetiology

There are five well recognized causes of auricular fibrillation, in a sixth group of cases the cause is not known In young subjects fibrillation may rarely appear in the course of a severe generalized infection.

Mitral stenosis—This is the commonest cause. When heart failure symptoms commence to appear in mitral stenosis, the heart rhythm may still remain normal, but when they have lasted for some time and have progressed, auricular fibrillation usually supervenes. When aortic incompetence, accompanying mitral stenosis, is the more severe heart lesion, normal rhythm is usually preserved in the face of heart failure.

Hypertension—Although heart failure in hypertension is more commonly associated with normal heart rhythm, auricular fibrillation is sometimes present It is less common in the hypertension accompanying nephritis, it is in simple hypertension that the arrhythmia is more common Auricular fibrillation does not materially affect the course and outlook of hypertensive heart failure because the addition of the arrhythmia does not always ensure a better response to digitalis.

Thyroid toxaemia—Auricular fibrillation is common in older subjects in whom a goitre has given rise to toxic effects. The arrhythmia is associated with a

varying degree of heart failure; in those patients exhibiting paroxysmal auricular fibrillation failure is minimal or even absent. If heart failure complicates thyroid toxaemia auricular fibrillation is a feature of the case. Digitalis will reduce the heart rate more readily than it does in hypertensive auricular fibrillation.

Cardiac infarction—Only a small number of patients with cardiac infarction develop auricular fibrillation. It usually happens early in the illness, lasting only for a short period, and it is rarely associated with the severe heart failure which may appear as a late complication of the disease, even though hypertension is present.

Constrictive pericarditis—Auricular fibrillation is common in constrictive pericarditis, even in young subjects. Indeed, in young patients with right heart failure of obscure origin, the presence of fibrillation should cause a search to be made for the signs of constrictive pericarditis.

Lone auricular fibrillation—In a group of patients none of the five causes already cited is operative Certain distinctive features characterize this type of fibrillation It is found in older but not necessarily elderly subjects, symptoms of heart failure are absent, the heart rate is not, as a rule, rapid as in other types, cardiac enlargement is absent, the outlook is satisfactory, and it yields readily to digitalis sufficient to lower the heart rate and cause the palpitation which may be present to disappear.

Symptoms and diagnosis

The diagnosis of auricular fibrillation depends on the following findings:

1 The pulse is *irregular*, except when complete heart block is present, and the irregularity persists in the face of tachycardia induced by exercise

2. The pulse is usually *rapid*, but it may be slow from digitalis therapy, in the presence of heart block, fibrillation of the lone type, thyrogenic fibrillation persisting after subtotal thyroidectomy, or when mitral stenosis shows gross cardiac enlargement with aneurysmal dilatation of the left auricle

3 The symptoms and signs of *heart failure* are usually present unless there has been adequate treatment, or when the fibrillation is of the lone type, or persisting in a patient with thyroid toxaemia after the operation of subtotal thyroidectomy.

4 With the exception of the lone variety, the *causative lesion* will exhibit its own peculiar signs.

5. A *jugular tracing* will show absence of the "a" wave, and distinctive features will show in the *electrocardiogram* Thus, the P waves are replaced by oscillations or "f" waves, these are best seen in the right pectoral lead (CR_1) (Figs. 78 and 79). The average rate of the auricular action is 400 per minute. The ventricular complexes may be normal except that they recur at irregular intervals, unless heart block is present. Under the effects of digitalis the variation in the intervals separating ventricular complexes may not be apparent, short of actual measurement. Again, under the influence of digitalis the ventricular complexes may become paired (coupling)

Auricular fibrillation is sometimes simulated by frequent extrasystoles, but should induced tachycardia convert the arrhythmia into normal rhythm the presence of extrasystoles is confirmed.

FIG. 78 —Auricular fibrillation in which fibrillation "f" waves are indistinct in limb leads, but are prominent in CR_1, and are indicated by dots

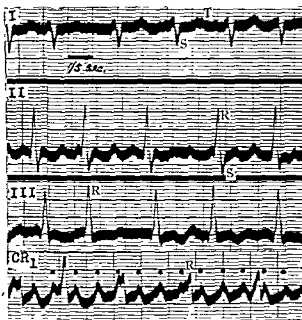


FIG 79 —Auricular fibrillation in which "f" waves are indistinct in limb leads, but are prominent in CR_1, and are indicated by dots

The *electrocardiogram* (Fig. 80) is characterized by a succession of irregular and often large waves unequally spaced. No auricular waves are depicted in the tracing

The outlook in ventricular fibrillation is necessarily grave, but in the transient type associated with Stokes-Adams disease the patient may survive repeated episodes of the arrhythmia.

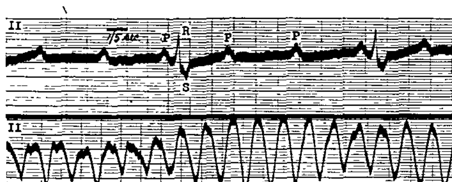


FIG 80—Ventricular fibrillation in the second tracing was associated with a Stokes-Adams attack The patient's usual rhythm of complete heart block is shown in the first cardiogram

Treatment is of little avail and for the most part should be directed to the associated heart lesion. Quinidine may sometimes inhibit or abbreviate the attacks, but it should be used cautiously in cases with complete heart block

FAULTY CONDUCTION OF CARDIAC IMPULSE

DELAYED AURICULO-VENTRICULAR CONDUCTION (LONG P–R PERIOD)

A delay in the conduction of the cardiac impulse from auricle to ventricle is regarded as present when the P–R interval in the electrocardiogram exceeds 0 22 second Such an arbitrary definition has inevitably meant that a P–R period slightly in excess of the normal is sometimes met with in healthy subjects, when it might alter with posture, lengthening with recumbency and shortening when the upright posture is assumed.

Aetiology

Occasionally the abnormality is found in congenital heart disease, especially when defect of the auricular or ventricular septa is present Rheumatic fever provides a common cause in young subjects when the condition may be temporary, although it is also found when mitral stenosis is already established. The specific infectious fevers, notably diphtheria, may delay for a time the cardiac impulse traversing auricle to ventricle when it is commonly associated with a block in one or both branches of the bundle of His Similarly, streptococcal septicaemia may prolong the P–R period in the electrocardiogram In older patients the condition, often associated with bundle branch block either in cardiac infarction or hypertension, is a lasting one.

Treatment

The treatment of auricular fibrillation has to be considered in relation to its cause and to the degree of heart failure accompanying it, and this is discussed in greater detail elsewhere (*see* p. 71).

In auricular fibrillation with mitral stenosis, digitalis gives the best results. Should rapid digitalisation be necessary it is best gained by giving 2 or 3 mg of digoxin by mouth. To maintain adequate digitalisation a choice of several preparations is available, but powdered digitalis leaf is both inexpensive and efficient. The single dose may be 1 grain, but the daily dose should be determined in each patient by clinical trial If oedema persists mercurial diuretics have to be used *in addition to digitalis*

In hypertensive auricular fibrillation, digitalis has distinct value, and hardly less than in the previous group of patients, but mercurial diuretics, after premedication with ammonium chloride, assume an important place in treatment.

Subtotal thyroidectomy is the appropriate remedy in thyrogenic auricular fibrillation, and by this means normal rhythm is re-established in the majority of patients In those cases in which this does not take place, quinidine needs to be prescribed, but not before the tenth day of the post-operative period. In a small proportion of cases the arrhythmia will continue in spite of quinidine therapy and, in these, quinidine should be given another trial some months later. Should auricular fibrillation persist after a successful operation it is unlikely to prove harmful even in the absence of treatment, although a small maintenance dose of digitalis may prove advantageous in that it will annul palpitation if tachycardia happens to be a feature. The presence of auricular fibrillation and heart failure does not contra-indicate subtotal thyroidectomy, but adequate treatment with digitalis and mercurial diuretics should be given before the operation takes place In these patients digitalis should be withdrawn as a rule on the fourth day after operation in order to facilitate a return to normal rhythm.

The lone type of auricular fibrillation may not require treatment, although a lowered heart rate by digitalis will ensure freedom from palpitation if tachycardia is present The operation of cardiac decompression will seldom reinstate normal rhythm in constrictive pericarditis, but continuous light digitalisation will prove satisfactory.

When auricular fibrillation sets in as a complication of cardiac infarction, digitalis should not be withheld in the fear that it may produce ventricular fibrillation.

In auricular fibrillation of young subjects, treatment must be directed to the infection causing it Digitalis will seldom be needed in these patients and quinidine should on no account be given.

VENTRICULAR FIBRILLATION

It is likely that ventricular fibrillation is fairly common during brief periods which immediately precede sudden death, especially during the post-operative crisis in thyroid toxaemia, and after cardiac infarction. Ventricular fibrillation has greater clinical significance when it is associated with Stokes-Adams attacks in a patient suffering from heart block. Here the arrhythmia precedes, follows, or occurs in the absence of, cardiac standstill.

The *electrocardiogram* (Fig. 80) is characterized by a succession of irregular and often large waves unequally spaced. No auricular waves are depicted in the tracing.

The outlook in ventricular fibrillation is necessarily grave, but in the transient type associated with Stokes-Adams disease the patient may survive repeated episodes of the arrhythmia.

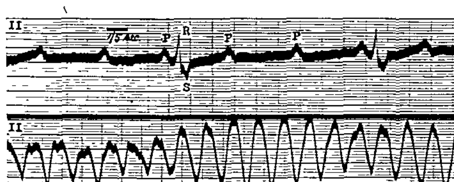


FIG 80—Ventricular fibrillation in the second tracing was associated with a Stokes-Adams attack The patient's usual rhythm of complete heart block is shown in the first cardiogram

Treatment is of little avail and for the most part should be directed to the associated heart lesion. Quinidine may sometimes inhibit or abbreviate the attacks, but it should be used cautiously in cases with complete heart block.

FAULTY CONDUCTION OF CARDIAC IMPULSE

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Aetiology

Occasionally the abnormality is found in congenital heart disease, especially when defect of the auricular or ventricular septa is present Rheumatic fever provides a common cause in young subjects when the condition may be temporary, although it is also found when mitral stenosis is already established The specific infectious fevers, notably diphtheria, may delay for a time the cardiac impulse traversing auricle to ventricle when it is commonly associated with a block in one or both branches of the bundle of His Similarly, streptococcal septicaemia may prolong the P–R period in the electrocardiogram. In older patients the condition, often associated with bundle branch block either in cardiac infarction or hypertension, is a lasting one.

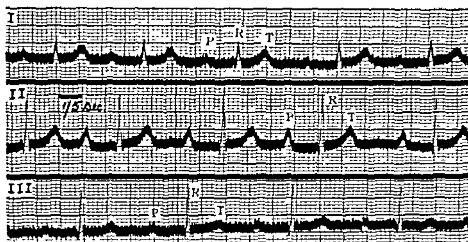


Fig 81 —Prolonged P–R period (0 3 second) in an otherwise healthy subject

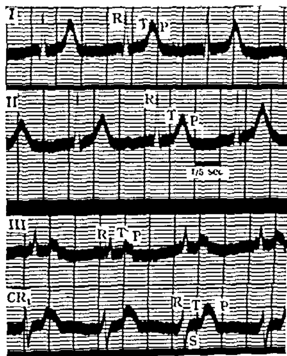


Fig 82 —The P–R period is so lengthened that the P and T waves coincide. Such fusion is usually best recognized in the chest lead CR_1 The cardiogram from a male aged 60 years subject to Stokes-Adams attacks

Symptoms and diagnosis

There are no subjective symptoms which can be assigned to this type of partial heart block and those which appear are common to the disease initiating the lesion A single objective sign, however, makes it possible to recognize it during clinical examination ; this sign is triple rhythm from addition of the fourth heart sound produced by auricular contraction. In young subjects such a finding is pathognomonic of a prolonged P–R period, but in adults hypertensive heart failure has *first to be excluded as a cause* *A prolonged P–R period does* not always initiate

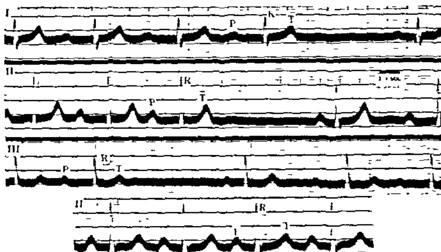


FIG 83—Sino-auricular block in a patient whose cardiogram customarily showed a very long P–R period This period is shortened in the beat which succeeds the silent gap, and after an *injection of atropine (lead II below) which produces a more lasting effect and annuls the* block temporarily

this triple rhythm for auricular contraction is often inaudible in adults, but as a rule it is easily heard in young subjects.

Confirmation of the diagnosis rests with the electrocardiogram, which will show the P–R period (measured from the commencement of the P to the commencement of the R wave) to exceed 0 22 second (Fig 81) ; often it is much greater *and on this account, or from tachycardia, the P* wave may coincide with the T wave (Fig 82) and in subsequent tracings it may be possible to watch the progressive separation of the T and P waves When a prolonged P–R period is associated with a wide ventricular complex exhibiting bundle branch block, in many cases it may be only slightly in excess of the normal. It is a feature of the electrocardiogram of myotonia atrophica.

Prognosis and treatment

A prolonged P–R period appearing during an acute illness is a sign of damage to the heart muscle, and this by itself should determine a resting period of at least two months for the patient. When this precaution has been taken, the prognosis will depend entirely on the causative lesion and it is gravest in diphtheria and

streptococcal septicaemia. The outlook is best in older patients, in whom the condition may appear over many years, but here again it depends on the associated cardiac defect which carries its own prognosis.

Apart from the resting period suggested for the condition when it arises during an acute illness, no specific treatment is known to be of value. Atropine can *shorten the P–R period* (*Fig. 83*), *but this has no place in practical therapeutics.*

ACCELERATED AURICULO-VENTRICULAR CONDUCTION (SHORT P–R PERIOD)

There has been assembled a group of cases which show a short P–R interval, ventricular complexes of bundle branch block type, and paroxysmal tachycardia (Wolff-Parkinson-White syndrome). The syndrome is found in about 5 per cent

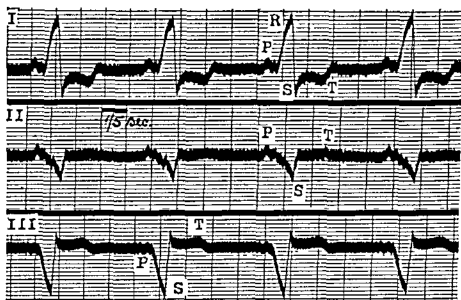


FIG 84 —The Wolff-Parkinson-White syndrome Short P–R period and bundle branch (left) block in a patient subject to paroxysmal tachycardia

of all cases of bundle branch block, and in the same proportion of patients subject to paroxysmal tachycardia. The characteristic *electrocardiogram* (Fig. 84) may be discovered during a routine examination in persons otherwise healthy, in patients *with paroxysms of tachycardia, or in those with some form of heart* disease. The prognosis seems to be unaffected by the addition of the syndrome even in a patient with associated heart disease. A special feature of the condition is that the same patient may at one time show the typical cardiogram and at another a normal one, both at normal rates, such a change may be spontaneous, though it may also be induced by exertion or by atropine. This temporary disappearance, and its natural disappearance with age, make it unlikely that the mechanism of the syndrome is concerned with an accessory bundle connecting auricle and ventricle. Further, multiple chest leads have failed to show early *excitation* of one or other ventricle, and histological examination has not always demonstrated an accessory muscular band.

INCOMPLETE AURICULO-VENTRICULAR BLOCK

Incomplete heart block is not a common event. Its causes are the same as those operating in complete heart block and they will be described under that section.

Symptoms and diagnosis

Palpitation spoken of by the patient as "the heart missing a beat" is a frequent symptom although by no means pathognomonic, because extrasystoles provide a commoner explanation of it. If other symptoms appear they are related to associated conditions such as hypertension or heart failure Sometimes the *block changes into the complete form and Stokes-Adams attacks may appear.*

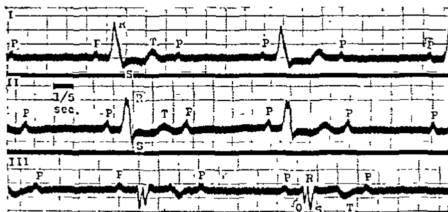


FIG 85—Heart block with 2 to 1 A–V dissociation The QRS complexes are very wide

The pulse is intermittent due to occasional "dropped beats" when the heart is silent on auscultation unless auricular systole is audible. The arrhythmia persists during tachycardia induced by exercise. It is noteworthy, however, that the clinical diagnosis of incomplete heart block is difficult and it is left to electrocardiography to interpret the true nature of the rhythm. In the tracing the form of both auricular and ventricular *complexes may be normal but the* sequence is irregular. In one variety (Fig 85) the ventricular complex of alternate beats fails to appear in response to auricular contraction. In the other variety (Fig 86) the P–R period lengthens in successive systoles until an expected ventricular complex fails to follow the auricular or P wave. The P–R period is shortest *after the pause and it lengthens progressively until the* next interruption. The increase in the second over the first interval is usually greater than the increase in the third over the second. Such phases are known as Wenckebach periods

The prognosis and treatment of incomplete heart block are those connected with the associated heart lesion, and are no different from those described under complete heart block.

COMPLETE AURICULO-VENTRICULAR BLOCK

When there is dissociation between auricular and ventricular systoles a state of complete heart block exists. The auricle continues to contract at the customary

rate in response to impulses arising in the sino-auricular node, but ventricular systole follows impulses arising within a newer station (the idioventricular node); its rate is much slower, usually 25-35 per minute, but occasionally, and especially in young subjects, it is more rapid (40-60). The condition sometimes appears in paroxysms, although more often it is established from the commencement.

Aetiology

In a small number of children with complete heart block the arrhythmia is

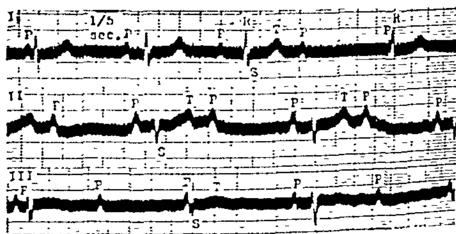


FIG. 86 – Heart block in which the P-R period in lead I lengthens successively in the first three beats until a ventricular beat fails to appear (Wenckebach periods)

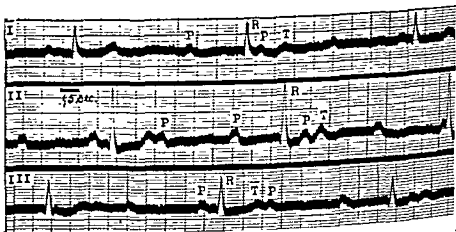


FIG 87 —Complete heart block Ventricular beats (33 a minute) independent of auricular beats (85 a minute)

congenital in origin when a defect of either the ventricular or auricular septum is often present.

Acute rheumatism is the commonest cause of complete heart block in children when mitral stenosis is a frequent associated lesion.

As a rule when recovery from *diphtheria* takes place, none of its effects on the heart persists even though cardiographic evidence of injury to the heart was present at the time of illness. Rarely, however, heart block developing during the acute phase of diphtheria continues during the convalescent period and remains as a permanent but trivial cardiac deformity.

Atheroma of the coronary leading to cardiac ischaemia or even to cardiac infarction, is the commonest cause of injury to the main stem of the bundle of His resulting in complete heart block.

Occlusion of the coronary orifice by *syphilitic aortitis* producing ischaemic changes is an uncommon cause of complete heart block, although commoner than actual gummatous infiltration of the bundle.

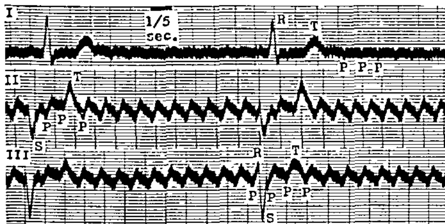


FIG 88—Complete heart block and auricular tachycardia Ventricular rate 26 a minute, auricular rate 364 a minute

Neoplastic deposits within the bundle have sometimes caused progressive interruption of the cardiac impulse

Complete heart block may be found amongst the electrocardiographic changes taking place in *Friedreich disease*, and in *myotonia atrophica*

Symptoms and diagnosis

Sometimes a patient may notice the bradycardia which characterizes complete heart block. Dyspnoea, heralding the onset of heart failure, may be the first symptom At the slower heart rate faintness may occur and this may give place to attacks of unconsciousness (Stokes-Adams disease). Other symptoms are an expression of the disease initiating the arrhythmia

The pulse is regular and slow (usually under 35 per minute), but sometimes, especially in children, the idioventricular impulses form more rapidly. The rate in heart block is hardly increased by exertion as in sinus bradycardia. The blood pressure is often raised. It may be possible to observe that the jugular pulsation (auricular systole) is about twice as rapid as the radial pulse (ventricular systole). The apex beat is often displaced outwards. Sometimes the separate auricular

systole can be heard on auscultation, and a mitral systolic murmur is a common finding when cardiac enlargement is considerable.

The *electrocardiogram* (Fig. 87) is distinctive, showing complete dissociation between the auricular wave and the ventricular complex, so that two independent forms of rhythm operate; the auricular rhythm is regular from 70 to 120 per minute, and the ventricular, usually from 25 to 35. The P waves have a normal form

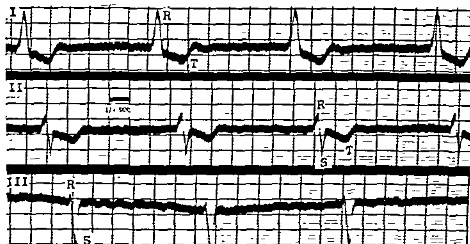


Fig 89—Complete heart block and auricular fibrillation The T wave is inverted in leads I II and IVR

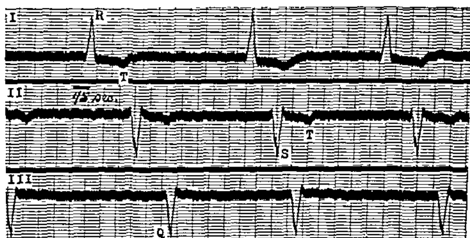


Fig 90—Great ventricular slowing (about 33 a minute) in auricular fibrillation simulating complete heart block; it is distinguished from it by the irregular ventricular rhythm. From a male aged 73 years with cardiac infarction.

unless auricular tachycardia (Fig. 88) or auricular fibrillation (Figs. 89 and 90) is an added feature. The P wave may be superimposed on either QRS complex or T wave. The QRS is often wide and the characteristic tracing of bundle branch block is frequently exhibited.

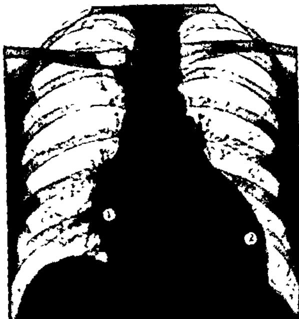


FIG. 91—Generalized enlargement of the heart, especially of the right auricle (1) and left ventricle (2), in a patient with complete heart block and slow ventricular rate (30 a minute)



FIG 92—Complete heart block, with a relatively high ventricular rate (80) had been present for some years and had not produced any enlargement of the heart in a male aged 63 years

Cardioscopy will usually demonstrate some degree of cardiac enlargement involving the left ventricle (especially in the presence of hypertension) and the right auricle (Fig. 91). Should the ventricular rate in heart block be faster than usual, enlargement of the heart is absent (Fig. 92). Hilar congestion may be present from heart failure A watch on the cardiac movements in the anterior view will often show a large excursion of the systolic stroke and a more rapid contraction of auricle than ventricle.

THE STOKES-ADAMS ATTACK

This terminology is applied to a period of unconsciousness from ventricular standstill, ventricular tachycardia, ventricular fibrillation, or a combination of these, which may take place in a patient with heart block. The auricle as a rule continues to beat during ventricular standstill.

Clinical features

Only about one-half the cases of complete heart block suffer from Stokes-Adams attacks, but the incidence in partial heart block is somewhat greater. Sometimes

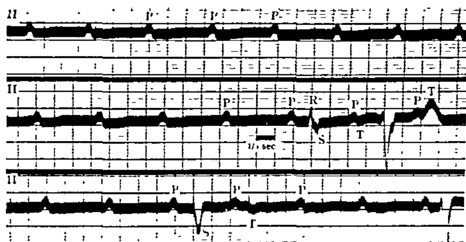


FIG 93 —Ventricular standstill No ventricular beats in the first tracing, two in the second, and two in the third

the attacks are so brief that a patient may complain of momentary giddiness and sway, but recovers control before falling. In such a patient it is usual to find the heart rate changing, and the bradycardia may only be moderate. Another patient may fall suddenly to the ground without warning, but recover consciousness quickly. Yet another patient may have warning of the attack, usually in the form of tachycardia, and utter a cry as unconsciousness sets in. The pulse stops and the heart ceases to beat (auscultation), but venous pulsation in the neck may still be visible. Sometimes a period of noticeable tachycardia precedes or follows the ventricular standstill. The breathing stops and as cyanosis develops, twitching of the face and convulsive movements of the limbs take place. After a variable time (even 10 seconds or longer) the pulse returns. Respiration re-appears, at first stertorous with heaving movements of the chest, and gradually

cyanosis gives way to pallor as consciousness is regained. Urinary incontinence is common when the attack is long, and the patient emerges confused and exhausted

Electrocardiographic features

When the mechanism of the Stokes-Adams attack is studied cardiographically during the period of unconsciousness, four different types may be recognized.

Type I. Ventricular standstill alone (Fig. 93).
Type II. (*a*) Low ventricular tachycardia ; (*b*) high ventricular tachycardia and fibrillation ; both kinds are followed by ventricular standstill.
Type III. Ventricular tachycardia and fibrillation without ventricular standstill
Type IV. Extreme bradycardia in heart block.

It is evident that ventricular standstill alone is not the only cardiac lapse that determines a Stokes-Adams attack ; it is often due to ill-action, not to in-action, of the ventricle Ventricular standstill is responsible for about 55 per cent ; ventricular tachycardia (with or without ventricular fibrillation) followed by ventricular standstill for 25 per cent, and ventricular tachycardia without standstill for 20 per cent. The mechanism described under *Type IV* is rare.

Prognosis

Assessing prognosis in complete as in other types of heart block, involves a consideration of the nature of the causative lesion. the presence of Stokes-Adams attacks, and the degree of cardiac enlargement or failure which may be present. In patients with Stokes-Adams disease, frequent attacks may give way to periods of variable duration, often years, when attacks are few or do not recur.

Treatment

Naturally, removal of the cause should be the first consideration, but this is seldom possible, unless in the uncommon instances when syphilis has provided the aetiological factor Among antisyphilitic remedies employed there should be a place for a mixture containing five grains of potassium iodide and one drachm of liquor hydrargyri perchloridi given three times a day for a period of not less than nine months If acute rheumatism, diphtheria, or other acute infections have caused the block, they should determine the patient's stay in bed for at least two months Similarly, if heart failure is present the patient should be rested till oedema and breathlessness lessen following the use of digitalis and mercurial diuretics Digitalis is helpful even in cases suffering from Stokes-Adams attacks because the drug is without much action on the idioventricular node.

In the main, treatment of heart block concerns the relief and prevention of Stokes-Adams attacks which feature so commonly in the course of the illness. Barium chloride, atropine sulphate, and thyroid have all been tried with the object of increasing the heart rate and preventing attacks, but seldom with satisfactory results. Adrenaline hydrochloride subcutaneously in doses of from 5 to 15 minims of 1 in 1,000 solution has proved to be the best therapeutic agent to use during the attack, for it may quickly raise the slow heart rate to about 40 per minute or higher, and the previous rate is only regained after a varying period (Fig. 94). Frequently, however, adrenaline fails to produce this favourable effect In many

patients half a grain of ephedrine given three times daily will maintain the heart at a slightly higher rate than the basic, providing immunity from attacks for long periods (Fig. 95) *The same improvement may often take place spontaneously and irrespective of any medicinal treatment.*

BUNDLE BRANCH BLOCK

Defective conduction in either of the two main branches of the bundle of His, constituting bundle branch block, is sometimes a transient lesion when it is associated with a short P–R period and attacks of paroxysmal tachycardia ; more

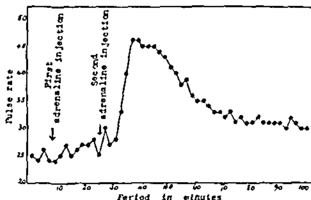


Fig 94 —Increase in the heart rate in a patient with heart block after the administration of adrenaline The response to the first injection of 10 minims of 1 in 1,000 solution was slight, but it was noticeable following a second injection 15 minutes later , this is common experience

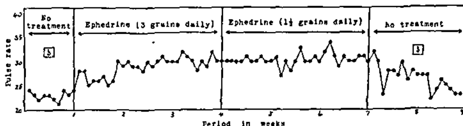


Fig. 95 —Increased heart rate and freedom from Stokes-Adams attacks during ephedrine therapy in a patient with complete heart block Figures in squares denote number of attacks during indicated periods.

often the defect is a lasting one. Frequently there is evidence of faulty conduction in the main bundle as well.

Aetiology

The causes mentioned under complete auriculo-ventricular block also operate in the case of bundle branch block. Thus, it may be found in congenital heart disease, especially in auricular or ventricular septal defect, diphtheria, cardiac ischaemia and infarction, and sometimes in Friedreich disease. It is seldom met with in acute rheumatism. Sometimes the bundle and its branches are subject to pathological changes ending in fibrosis when the arteries supplying the bundle are healthy.

Symptoms and diagnosis

The symptoms of bundle branch block are directly related to the clinical condition causing it. Splitting of the first or second heart sounds is a common auscultatory sign and a triple rhythm from addition of the fourth heart sound may be heard, although the wide QRS period causes separation of the auricular and ventricular portions of the first sound (splitting), triple rhythm is not heard unless the P–R period is also prolonged, or left ventricular failure is present.

As in complete heart block some degree of hypertension is common and so is moderate cardiac enlargement, which is only evident on cardioscopy. In left

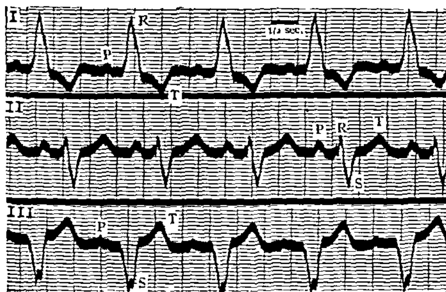


FIG. 96.—Left bundle branch block.

bundle branch block the left ventricle is commonly enlarged from hypertension, and in right bundle branch block the right ventricle may sometimes be relatively greater, but such relationship is by no means constant.

The recognition of the particular branch of the bundle which has been damaged rests with the *electrocardiogram*, but even this can only supply part of the truth Thus, while a characteristic tracing has been held to typify an injury to each branch, histological examination usually demonstrates involvement of both branches, although one is more affected than the other according to the pattern preponderating in the tracing. The *cardiogram* which characterizes a bundle branch block shows a wide QRS complex, usually much wider than the normal (0·1 second) and often exhibiting a larger voltage, with notching of its ascending or descending limb. The T waves (known as secondary T waves) are written in the opposite direction to the QRS deflection, but not necessarily in all leads The P–R period is commonly prolonged

In the electrocardiogram denoting a preponderating lesion of the left branch (Fig. 96), the QRS complex is directed upwards in lead I, downwards in lead III, and variably in lead II.

In the electrocardiogram denoting a preponderating lesion of the right branch the QRS complex is directed downwards in lead I, upwards in lead III, and variably in lead II. This pattern, which is the counterpart of the one depicting left bundle branch block, is, however, the uncommon curve (Fig 97) The common curve (Fig. 98) indicating right bundle branch block is characterized

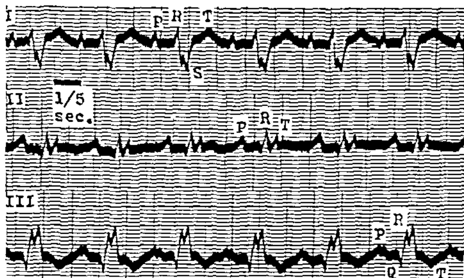


FIG 97.—Right bundle branch block (uncommon curve)

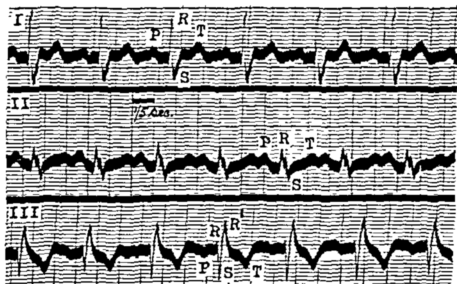


FIG 98.—Right bundle branch block (common curve)

by a deep S wave as a component of a wide QRS complex in lead I and also in lead II as a rule. The T wave is upright in leads I and II and is usually inverted in lead III. In CR_2 the main QRS deflection is upright and the T wave is inverted.

Attention has already been directed to the variety of bundle branch block which is associated with a short P–R period and found in patients susceptible to attacks of paroxysmal tachycardia, and to the common incidence of complete heart block as an occasional or permanent finding in bundle branch block.

Prognosis and Treatment

The outlook in this condition depends on the lesion causing it. Its presence in diphtheria or streptococcal septicaemia is an ominous sign, for it tells of myocardial involvement. It is immaterial in young subjects with congenital heart disease. In elderly subjects the condition is compatible with good health provided it has not issued as the result of cardiac infarction.

Treatment of patients with bundle branch block must necessarily be directed to the disease initiating the lesion, so that it must concern diphtheria, septicaemia, hypertension, heart failure, complete heart block, paroxysmal tachycardia, and rarely syphilis

FAULTY CARDIAC CONTRACTION (PULSUS ALTERNANS)

There is experimental evidence that alternation in the heart action results from a relative failure of the whole or part of the heart to contract normally during alternate beats.

Aetiology

Pulsus alternans is not uncommon in conditions associated with a rapid heart action, especially paroxysmal tachycardia, in which it is of no significance. Heart failure in simple or papilloedemic hypertension supplies the commonest cause of pulsus alternans. It may also appear in cardiac infarction either at the onset of the illness or later when heart failure or cardiac aneurysm has resulted. Aortic incompetence, especially the syphilitic form, is another cause of pulsus alternans, when left ventricular heart failure is usually present.

Symptoms and diagnosis

Symptoms cannot be attributed directly to pulsus alternans and they are the outcome of the disease producing the abnormal condition. It is often possible to discover alternation of the radial pulse by palpation, and in those not discovered by this means it may be recorded by an arteriograph, in such a tracing the height of the taller wave is fairly constant but the alternating shorter wave varies in amplitude Pulsus alternans is most readily made out by the sphygmomanometer. As the cuff pressure is lowered, and at the point marking the systolic blood pressure, only half the beats are audible over the brachial artery. When the pressure is lowered further the remaining beats become audible so that the original pulse rate is doubled. The measure of the alternation may be expressed as the difference between the two levels in terms of mm. of Hg. Thus, in a patient with hypertensive heart failure the initial systolic blood pressure reading was 220 and the pulse rate was 45 per minute; when the cuff pressure was reduced slowly

to 215 the rate was doubled so that it showed an alternation of 5 mm. of Hg recorded as 220-215 (p.a.)/120. Only rarely is this variation seen in the electrocardiogram, and on the other hand the majority of cases showing electrical alternans do not exhibit pulsus alternans.

Pulsus alternans needs to be distinguished from alternating extrasystoles in which the alternate premature beats at the wrist are less forcible. The true interpretation rests with the recognition of the compensatory pause which follows the smaller beat producing a coupling effect; this pause is not present in pulsus alternans. Confirmation of the diagnosis is obtained from the electrocardiogram (Fig. 99).

Prognosis

This physical sign must not be singled from amongst others which a patient may show and some sinister meaning apportioned to it. For instance, pulsus

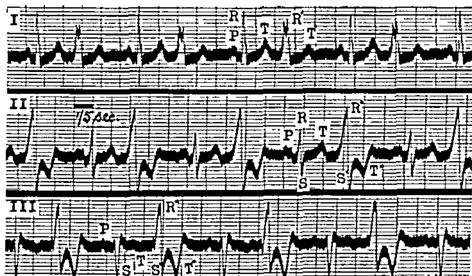


FIG. 99.—Alternating extrasystoles in which the compensatory pause is only a little longer than the period which precedes it, giving the impression of pulsus alternans when examining the pulse at the wrist; the cardiogram gives the proper interpretation

alternans in paroxysmal tachycardia is without significance. When found in association with conditions like cardiac infarction and hypertensive heart failure, it must not weigh too heavily in estimating prognosis, which should be judged largely in relation to the condition producing the irregular cardiac action.

Treatment

In treatment too, attention should be directed to the condition initiating the alternation, so that in paroxysmal tachycardia quinidine may play an important part, whereas in hypertensive heart failure, rest, mercurial diuretics and digitalis serve as the best therapeutic aids.

CHAPTER 5

CONGENITAL CARDIOVASCULAR DISEASE

In order to allot to congenital cardiovascular anomalies a more exact prognosis, it is necessary to recognize clinically the actual anatomical defects which are present. This precision in diagnosis is sometimes difficult especially when more than one congenital abnormality exists in the same patient. In the case of infants who show cyanosis from congenital heart disease, any attempt at a diagnosis of the defects which may be present would be unwise because of the bizarre abnormalities that might exist making of it a freakish case Our knowledge of congenital cardiovascular disease will only become complete when there is assembled a sufficient number of case records which include clinical, electrocardiographic, radiological, phonocardiographic, and pathological findings. Such records are already accumulating and they permit of dogmatic statements on the diagnosis of many combined congenital defects as well as single lesions in children and adults.

GENERAL SYMPTOMS

Certain symptoms are common to many congenital cardiovascular disorders and they will be considered first. The special symptoms associated with a particular congenital lesion will be described later under each condition.

DYSPNOEA

Shortness of breath on exertion is a common presenting symptom of congenital heart disease ; indeed, when this is noticed in a child, a congenital cardiovascular defect is the likely explanation. The symptom is the result of cyanosis due to entry of venous blood into the arterial circulation or, much less frequently, the outcome of heart failure which makes its appearance late in the illness. Thus, if the defect establishes a veno-arterial shunt and non-oxygenated blood flows into the arterial system, dyspnoea results and is associated with cyanosis. On the other hand, if the shunt is arterio-venous in type and oxygenated blood flows into the venous system, dyspnoea and cyanosis are absent unless the shunt is reversed by any condition which raises the venous pressure, such as exercise, incidental emphysema, pulmonary infarction, or heart failure.

CYANOSIS

The cyanosis of congenital heart disease is the result of insufficient oxygenation of the blood caused by a veno-arterial shunt. It is estimated that about one-third of the venous blood must enter the arterial system before a state of cyanosis is established. Examination of the blood shows a high red cell count, high haemoglobin content, and a low oxygen-saturation value. In patients with congenital heart disease who exhibit a low grade cyanosis, this symptom may become obvious only after exercise (effort or episodal cyanosis). Maude Abbott, who contributed greatly to our knowledge of congenital heart disease, suggested a classification

for these anomalies based on the presence and prominence of cyanosis. This classification is followed here except that some of the conditions have been excluded on the grounds that they are unimportant in clinical medicine in that patients suffering from them seldom survive infancy.

GROUP I. WITHOUT CYANOSIS (NO ARTERIO-VENOUS COMMUNICATION)

Dextrocardia
Bicuspid aortic valve
Coarctation of the aorta (closed ductus)
Aortic and subaortic stenosis

GROUP II. WITH EPISODAL CYANOSIS (ARTERIO-VENOUS SHUNT)

Auricular septal defect
Ventricular septal defect
Patent ductus arteriosus
Coarctation of the aorta (open ductus)

GROUP III. WITH CYANOSIS (VENO-ARTERIAL SHUNT)

Pulmonary stenosis (complicated)
Pulmonary atresia (complicated)
Fallot's syndrome
Right-sided aortic arch

ARTHROPATHY

Clubbing of fingers, toes, and sometimes of the nose in congenital heart disease usually progresses in association with the cyanosis which induces it. Moderate cyanosis may be present without clubbing, especially in infants, but whenever clubbing is found in congenital heart disease, cyanosis is always a feature It is a valuable sign in diagnosis for, with rare exceptions, it may be said that provided bacterial endocarditis is excluded, clubbing of the fingers in a patient with heart disease defines the cardiac defect as a congenital one.

CEREBRAL ATTACKS

Attacks of faintness and pallor may feature in some instances of congenital heart disease, particularly aortic stenosis and patent ductus arteriosus Convulsions and unconsciousness occur in infants when cyanosis is conspicuous Hemiplegia may result from cerebral thrombosis in patients with extreme cyanosis as in the case of pulmonary atresia Cerebral symptoms due to a brain abscess have sometimes been described, but that this is a rare event is emphasized by the absence of congenital heart disease in 197 consecutive cases of brain abscess examined at necropsy.

MURMURS

The murmurs are mainly systolic in time and are commonly situated in the pulmonary area ; diastolic murmurs are present in patent ductus arteriosus and auricular septal defect. Often the murmurs are rough in character and are frequently accompanied by thrills The lesser defects may produce the loudest murmurs, whereas in a severe deformity such as pulmonary atresia there

may be no murmur. The pulmonary second sound is accentuated in many examples of congenital heart disease, but it does not constitute a sign of diagnostic importance.

ELECTROCARDIOGRAPHY

The specific electrocardiogram for the various congenital lesions will be described later and the following remarks apply only in a general way to the tracing found in this group of heart diseases. The tracing commonly shows a high voltage, deviation of the electrical axis to the right, bundle branch block, and inversion of the T wave or P wave in one or more leads. Rarely, complete heart block is present. So characteristic are these changes that examination of a patient with congenital heart disease is incomplete without electrocardiography.

CARDIOSCOPY

Cardioscopy has contributed in great measure to the precise diagnosis of congenital disease of the heart and greater vessels. As with electrocardiography, so also with cardioscopy, the investigation of a patient is incomplete without this test. In the anterior view special notice should be taken of the position of the aortic knuckle, the relation of the barium-filled oesophagus to the aortic arch, the pulmonary artery and its branches, the conus of the right ventricle, and the right auricle. In the right oblique position the conus and the pulmonary artery and its right branch are well seen In the left oblique position the body of the right ventricle and the left pulmonary artery should be specially examined.

PROGNOSIS

The outlook in a particular cardiovascular anomaly is not stereotyped and it depends on several factors such as the kind of defect, its severity, and the complications which may arise. The prognosis is moderately favourable up to young adult life in patent ductus arteriosus, even without operation, and in ventricular and auricular septal defects, provided these are small and have not caused great cardiac enlargement. The outlook in pulmonary atresia is improved by the presence of a ventricular septal defect. Pulmonary infection and, in the later stages, heart failure, are complications to which a patient with congenital heart disease is prone, but the susceptibility of most patients with congenital defects to bacterial endocarditis disturbs above all else an estimate of longevity in a particular patient.

TREATMENT

Although nothing spectacular can be achieved in the treatment of most cases of congenital heart disease, much may be gained by the thoughtful management of a case. A child in the acyanotic group is not likely to be breathless while at play and the exclusion of competitive games is all that is necessary in such cases In the cyanotic child breathlessness will naturally restrict activities and the patient should be exempted from compulsory routine school exercises and drill. Both parent and teacher should train and educate the child with congenital heart disease for a sedentary occupation. Parents planning for higher education connected with one of the professions may be informed of the average longevity associated with a particular congenital lesion if they specially seek this knowledge. Should pulmonary tuberculosis develop, as it does sometimes, it will demand

its own specific treatment. When bacterial endocarditis supervenes, penicillin treatment should be commenced immediately. The proper care of the teeth and penicillin therapy before dental extraction, in a patient with a congenital heart defect, may have some influence in preventing the onset of this fatal complication. When heart failure has set in as a terminal event, occasional venesection and the routine use of mercurial diuretics and digitalis will give the best results Surgical treatment has altered the outlook in patent ductus arteriosus and it is under trial in coarctation of the aorta and in pulmonary stenosis.

GROUP I WITHOUT CYANOSIS (No arterio-venous communication)

DEXTROCARDIA

Congenital dextrocardia in which the heart occupies more of the right compartment of the thorax with the apex beat pointing to the right, may present in two ways. In the first type there is transposition of the abdominal viscera as well as the

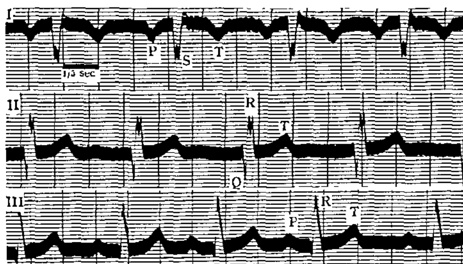


FIG. 100—Congenital dextrocardia The P, QRS, and T waves are inverted in lead I, the P wave in lead II is flat and the Q wave is deep

heart so that a mirror image of the normal is obtained. In the second and rare type the heart alone is transposed and it is then usually affected by other severe congenital abnormalities, so that the patients seldom survive infancy; in the majority of these cases a true mirrored effect is not produced.

SYMPTOMS AND DIAGNOSIS

The symptoms connected with the second type are severe and related to the associated congenital anomalies. Symptoms do not arise from the first type of congenital dextrocardia, but two abdominal conditions owe their peculiar symptoms to it. Thus, in acute appendicitis the generalized abdominal pain settles in the left iliac fossa, whereas in cholecystitis the pain is in the left hypochondrium and referred to the left shoulder. Indeed, the importance of congenital dextrocardia

belongs to the identification of transposed abdominal viscera when these call for surgical treatment.

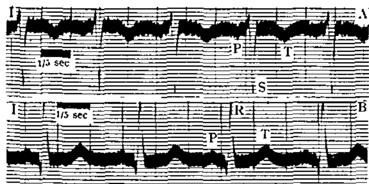


FIG 101—Congenital dextrocardia Inversion of the three primary waves in lead I (shown in A) is corrected (B) by transposing the arm leads.

FIG 102—Pulmonary stenosis, congenital dextrocardia, and congenital heart block Although the P and T waves are inverted in lead I, the QRS complex is directed upwards because of right axis deviation from pulmonary stenosis The lower lead I tracing, obtained by transposing the arm leads, explains the changes

The apex beat is found on the right side and the heart sounds are similarly disposed, together with murmurs should other congenital abnormalities be present.

The *electrocardiogram* in congenital dextrocardia is characteristic (Fig. 100). The three primary waves, P, QRS, and T, are inverted in lead I; the P wave

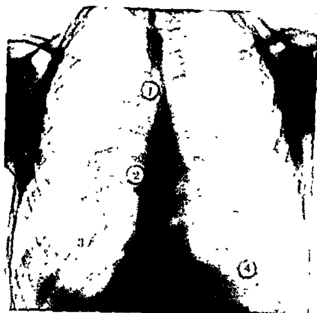


FIG 103.—Congenital dextrocardia Reversal of the aortic arch impression (1) and the left auricle impression (2) The stomach (3) is on the right, and the liver (4) is on the left

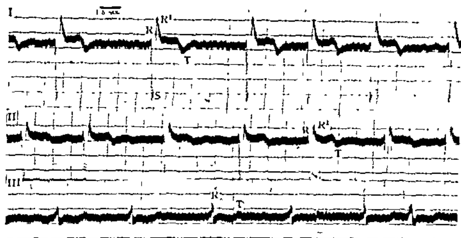


FIG 104.—Congenital dextrocardia and auricular fibrillation The S-T segment in leads I and II is raised (depressed in the normally placed heart) because of hypertension

may be flat or inverted in lead II as well. If doubt arises in the interpretation of the tracing obtained in a patient with dextrocardia, the electrocardiograph may be accommodated to the patient by transposing the arm leads and taking

the leg lead from the right instead of the left, the record obtained (Fig. 101) is a physiological one. Should a congenital defect, such as pulmonary stenosis, be present as well, the R wave is directed upwards in lead I as a mark of right axis deviation and the P and T waves remain inverted (Fig. 102).

Cardioscopy will demonstrate the transposed heart, stomach, and liver, and the impressions on the barium-filled oesophagus are reversed (Fig 103) The presence of other congenital defects will change the cardiac outline in a way characteristic of them.

PROGNOSIS

In the absence of any other congenital cardiac abnormalities the outlook in a *case of dextrocardia is no different to that for the normal heart.* It is subject to the same form of acquired disease, and the electrocardiogram shown in Fig 104 is from a patient with congenital dextrocardia, hypertension and auricular fibrillation, who died at the age of 72 from carcinoma of the uterus

BICUSPID AORTIC VALVE

This congenital defect assumes importance on account of its liability to develop pathological changes either in the form of thickening and calcification or, more frequently, the complication of bacterial endocarditis. this happens in some one-fifth of the cases. Supernumerary aortic cusps do not appear to predispose to the complication in the same way.

SYMPTOMS AND DIAGNOSIS

There is no means of recognizing the presence of a bicuspid aortic valve before complications have set in, and even then it cannot be identified with certainty If a patient with bacterial endocarditis and aortic stenosis showed no aortic valvular disease at a previous examination a congenital bicuspid aortic valve is likely to have been the basis of the infection.

COARCTATION OF THE AORTA (CLOSED DUCTUS)

A slight and gradual narrowing of the aortic isthmus is a normal finding during the first three months of post-natal life and the deformity disappears by the fourth month, but the pathological variety shows an abrupt narrowing (occasionally atresia) of the aortic lumen, persisting in adult life (Fig 105) The deformity is usually found at the level of the aortic attachment of the ductus arteriosus or just beyond, and, as a rule, distal to the left subclavicular artery. The condition has an incidence of 1 in 1,000 in routine necropsies.

ANATOMICAL TYPES

The effect of stenosis of the aortic arch on the heart and on the aorta and its branches lying proximal to the constriction, depends on whether the ductus arteriosus is open or closed. In cases in which the ductus is closed, there is hypertrophy of the left ventricle and of the aorta and its branches proximal to the site of stenosis. Actual atresia of the aorta is sometimes met with in this group

SYMPTOMS AND DIAGNOSIS

No single symptom can be regarded as characteristic of congenital stenosis of the aortic arch, and complaints arise from complications such as heart failure, rupture of the aortic arch, bacterial endocarditis or endarteritis, or cerebral haemorrhage. Some patients remain symptom-free. The objective symptoms are characteristic and readily permit a clinical diagnosis of the anomaly; they are conveniently considered in two groups, the one comprising symptoms common to hypertension and the other symptoms peculiar to coarctation hypertension.

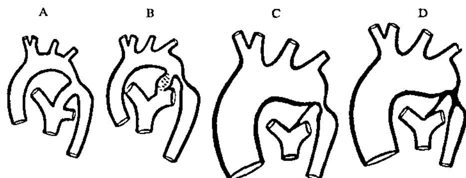


FIG 105—Anatomical features of the separate types of congenital stenosis of the aortic arch (coarctation) (A) Physiological (B) Coarctation with patent ductus (C) Coarctation with closed ductus (D) Atresia with closed ductus

Signs common to hypertension

As a rule there is increased and obvious pulsation in the radial, brachial and carotid arteries. The brachial blood pressure is usually raised to values like 230/120, but occasionally it may be as low as 150/90 The apex beat is displaced outwards *The second heart sound in the mitral and aortic areas is accentuated* The electrocardiogram may show left electrical axis deviation with left ventricular preponderance, and cardioscopy will demonstrate enlargement of the left ventricle.

Signs peculiar to coarctation hypertension

Rarely, the stenosis affects the aorta proximal to the origin of the left subclavian artery so that the radial pulse will be large on the right and small on the left.

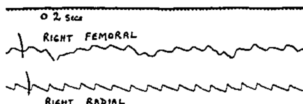


FIG. 106—Delay in the femoral pulse in coarctation of the aorta; it precedes the radial pulse in healthy subjects

The femoral pulse is usually absent to palpation and if present it is very small; the femoral blood pressure too is very low (for example, 90/0) In the normal subject the femoral pulse precedes the radial, but in coarctation the reverse is true, and synchronous radial and femoral pulse tracings will demonstrate this (Fig. 106).

The presence of tortuous hypertrophied arteries of the collateral circulation provides further evidence of coarctation. Thus, the internal mammary arteries may be seen in the intercostal spaces adjacent to the sternum (Fig. 107), and scapular arteries at the back (Fig. 108). These abnormal arteries may be felt if they are not seen, and a systolic murmur is heard over them ; the murmur is naturally in late systole (Fig. 109). If there is great enlargement of the heart it

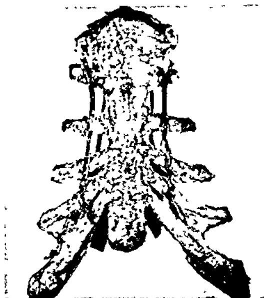


FIG 107—Enlarged internal mammary arteries from a case of coarctation of the aorta

may produce its own murmur in mid-systole An early diastolic murmur from relative aortic incompetence is not uncommon in coarctation hypertension Cardioscopy, in addition to showing enlargement of the left ventricle, will show absence of the aortic knuckle on the left and prominence of the ascending aorta to the right, in the anterior view, while in the left oblique position the aortic triangle is usually absent (Fig 110), this sign has great value because it can be recognized on cardioscopy, and it does not need a teleradiogram for its demonstration, as is the case with erosion of the lower borders of the ribs by tortuous hypertrophied intercostal arteries (Roesler's sign) (Fig. 111).

PROGNOSIS

Although patients with coarctation and a closed ductus may attain old age, the outlook is uncertain because of their susceptibility to bacterial endocarditis or endarteritis, rupture of the first part of the aorta, heart failure and cerebral haemorrhage. Although hypertension in the circulation proximal to the stenosis is a determining cause of the rupture, one of two other conditions will be found present ; either medial degeneration of the aorta or a mycotic aneurysm from bacterial endocarditis. The more severe the stenosis the greater, as a rule, is the

liability to complications, so that this feature indirectly influences the prognosis in individual cases. Although the severity of the stenosis cannot be accurately

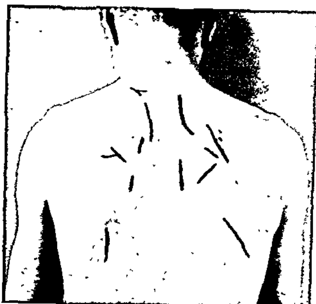


Fig 108.—Black lines indicate the site of pulsating hypertrophied arteries of the collateral circulation formed in a patient with coarctation of the aorta

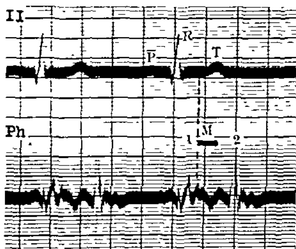


Fig 109.—A phonocardiogram from the mitral area in a case of coarctation of the aorta showing the murmur (M) in late systole

judged, an estimate may be made from the extent to which the collateral circulation has formed, the prominence of the ascending aorta, the degree of enlargement



Fig 110.—Coarctation of the aorta in a male aged 25 years Absence in the left oblique position of the aortic triangle of light (1).

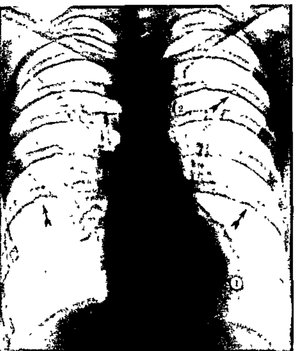


Fig 111.—Coarctation of the aorta in a male aged 25 years. The left ventricle (1) is enlarged, and there is absence of the aortic knuckle (2). Multiple rib erosions, some of which are indicated by arrows

of the left ventricle at cardioscopy, and the extent to which the inferior borders of the ribs are eroded.

TREATMENT

The onset of heart failure may be postponed by avoiding exertion while continuing to indulge in exercise. When heart failure or other complications occur, appropriate treatment should be given Surgery is under trial in the treatment of coarctation. Already in a few cases the constriction has been resected and the aorta joined end to end. Occasionally the operation does not abolish the hypertension and this suggests a renal origin for hypertension in coarctation

AORTIC AND SUBAORTIC STENOSIS

Two anatomical types may result from a developmental abnormality of the aortic orifice. In the first the narrowing is the outcome of thickening of the cusps, and in the second it takes place in the neck of the left ventricle just below the cusps, when it is due to a failure of the bulbus cordis to atrophy; in this type the cusps may also be thickened, the aorta may show hypoplasia, and the ventricular septum may remain patent.

SYMPTOMS AND DIAGNOSIS

If the aortic narrowing is substantial the general development is retarded and even dwarfism may appear Pallor and giddiness are sometimes present. The



FIG 112 —Congenital subaortic stenosis There is right axis deviation with wide QRS complexes

pulse is small and the systolic blood pressure low. The apex beat is displaced outwards a little and is forcible A rough systolic murmur in the aortic area is conducted into the vessels of the neck and is usually accompanied by a thrill The clinical features of subaortic stenosis resemble the acquired type of aortic stenosis, although in the congenital variety the obstruction is more severe at an earlier age, physical development is more likely to be impaired, the pulse and blood pressure are more appreciably altered, cardiac enlargement is more evident,

and the murmur may be more localized. Although the aortic second sound is always present this cannot help in differential diagnosis because it is commonly heard in acquired aortic stenosis.

The *electrocardiogram* may occasionally show right electrical axis deviation (Fig. 112) Sometimes the T wave is inverted as in the acquired type. The differential diagnosis of the congenital and acquired type is very difficult, and the finding of right axis deviation in the cardiogram, if it is present, reveals the congenital nature of the stenosis

Cardioscopy typically demonstrates enlargement of the left ventricle and prominence of the ascending aorta to the right. Calcium may be seen in the cusps.

PROGNOSIS

In a series of 23 cases the maximal age was 58 and the average 13 years. The outlook depends on the severity of the stenosis and the presence of complications, of which heart failure and bacterial endocarditis are the commonest, pulmonary tuberculosis is less frequently encountered

GROUP II. WITH EPISODAL CYANOSIS (Arterio-venous shunt)

AURICULAR SEPTAL DEFECT

A defect of the auricular septum is present in some 35 per cent of routine necropsies, but in 30 per cent it is small and only admits a probe. In about 5 per cent of cases it is large enough to admit a pencil. It is only when the opening

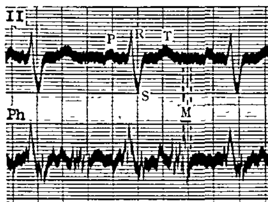


FIG 113 —Auricular septal defect. Phonocardiogram in mitral area showing early diastolic murmur of pulmonary incompetence ; the murmur starts after the second sound

is about the size of a halfpenny that changes in the heart result from this congenital defect. The actual changes that take place involve the right side of the heart, so that there is enlargement of the right auricle, right ventricle, and the pulmonary artery and its branches. There is no enlargement of the left auricle and left ventricle ; in fact the left ventricle and the aorta may appear smaller than is normal.

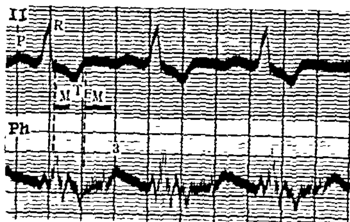


FIG 114—Auricular septal defect. The early diastolic murmur from pulmonary incompetence lasts up to a prominent third heart sound (3) The systolic murmur starts at the S line

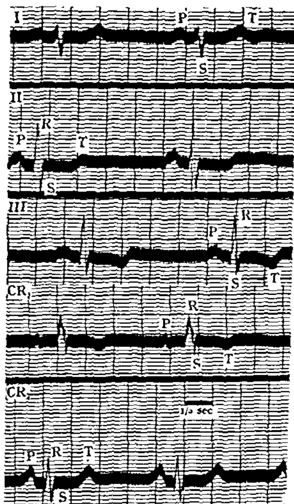


FIG 115—Auricular septal defect Right axis deviation T wave inverted in leads III and CR_1 T upright in CR_7 R-T depression in II S wave small in CR_1

SIGNS AND DIAGNOSIS

Although physical underdevelopment may occur in auricular septal defect it is not common. Cyanosis is late in its onset ; its appearance may imply the onset of heart failure or the association of mitral stenosis (Lutembacher's syndrome) Similarly dyspnoea is a late feature and so is arthropathy. The pulse is small and the systolic blood pressure is low. The apex beat may be displaced outwards a considerable distance due to enlargement of the right heart. A thrill may show in the pulmonary area The auscultatory signs are variable. A systolic murmur is commonly present in the mitral and pulmonary areas and is an expression of cardiac enlargement. An early diastolic murmur of pulmonary incompetence is heard in the pulmonary and mitral areas (Fig 113), and is a valuable physical sign, this murmur, which follows immediately after the second heart sound, is continued up to the distinct third heart sound, giving rise to a triple rhythm (Fig 114) Indeed, among all forms of congenital heart disease, auricular septal defect is the most certain to give rise to triple heart rhythm from the addition of the third heart sound

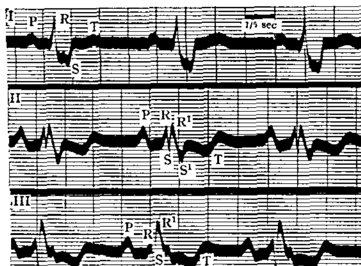


Fig 116—Auricular septal defect Electrocardiogram showing right bundle branch block

The *electrocardiogram* (Figs. 115 and 116) is usually abnormal and shows deviation of the electrical axis to the right, inversion of the T wave in leads II, III and CR_1 as an expression of right heart preponderance, or bundle branch block.

On *cardioscopy* (Figs. 117 and 118) there is generalized enlargement of the heart, chiefly to the left, the extension of the left border outwards is due, however, to an enlarged right ventricle which displaces the left ventricle. If enlargement of the right auricle is unusually conspicuous it suggests mitral stenosis as well. Prominence of the pulmonary arc and a dense shadow in the right hilum in the form of a " reversed comma " (right branch of the pulmonary artery), which usually shows excessive pulsation (hilar dance), gives an auricular septal defect

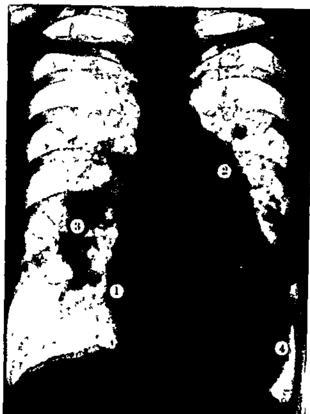


FIG 117.—Auricular septal defect Slight enlargement of the right auricle (1), and great prominence of the pulmonary artery stem (2) and of the right pulmonary artery (3) which showed great pulsation on cardioscopy (hilar dance) Left ventricle (4) is displaced to the left by the enlarged right heart



FIG 118.—Auricular septal defect Left ventricle (1) displaced to the left by the enlarged right heart Enlargement of the right auricle (2) and great enlargement of the pulmonary artery (3) and its branches (4) which showed great pulsation on cardioscopy (hilar dance) From a male aged 20 years

its characteristic appearance in the anterior view. The aortic knuckle is small. In the right oblique position the enlarged pulmonary arc is again evident and the left bronchus impression on the oesophagus is elongated In the left oblique view an enlarged left pulmonary artery obscuring the aortic window and a prominence of the right ventricle are two characteristic features.

PROGNOSIS

The age at death is usually from 30 to 50, and death results from heart failure, pulmonary infarction, paradoxical embolism and, very rarely, bacterial endocarditis.

PARADOXICAL EMBOLISM

A paradoxical or crossed embolism entails the passage of an embolus consisting of either a thrombus (Fig. 119) or neoplastic or septic material, through a defect



FIG 119.—Large paradoxical embolus seen in three of the heart cavities viewed from the back. The head of the embolus (1) is in the left ventricle, the main body (2) is in the left auricle, and the tail (3) is in the right auricle.

in the auricular or ventricular septum, into the left heart chambers and thereafter into the systemic circulation The cerebral artery is the commonest site of embolism although it may sometimes take place in the renal, splenic, coronary or peripheral arteries Paradoxical embolism accounts for 4 per cent of all the

cases of cerebral embolism. It is unlikely that pulmonary infarction initiates the condition by raising the blood pressure in the right side of the heart, in that it is necessary in the first place to deplete one-third of the pulmonary circulation *in order to raise the blood pressure in the right heart higher than in the left*; secondly, when one-half of the pulmonary circulation is depleted suddenly by embolism the patient dies; thirdly, even when the first condition pertains it is necessary for a further embolus to arrive in the right heart in order to pass to the left as a paradoxical embolism.

The condition may be recognized clinically with reasonable certainty. Thus, when systemic embolism has resulted in the course of venous thrombosis, and disease of the left ventricle and aorta can be excluded, the embolism is likely to be of the paradoxical type.

LUTEMBACHER'S SYNDROME

A combination of auricular septal defect and mitral stenosis makes up the syndrome. The effect of mitral stenosis on the heart already showing changes from the septal defect is to increase the enlargement of the right heart as well as to produce some enlargement of the left auricle.

SYMPTOMS AND DIAGNOSIS

When auricular septal defect and mitral stenosis have been associated for a number of years, *cyanosis sets in and increases. Breathlessness also appears,*

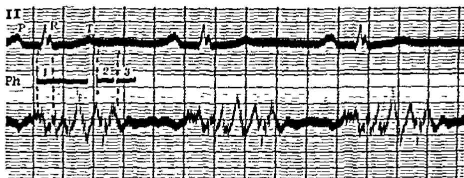


FIG 120—Auricular septal defect and mitral stenosis The systolic murmur commences during auricular systole (1) and within the P-R period An early diastolic murmur (2) from pulmonary incompetence follows the second heart sound, and a mid-diastolic murmur (3) from mitral stenosis follows the third heart sound

but in spite of this, together with conspicuous cardiac enlargement, anasarca is uncommon. Indeed, when an adult with mitral stenosis and gross cardiac enlargement develops cyanosis and breathlessness without oedema of the extremities, this dual lesion needs to be kept in mind as the possible explanation.

Auscultation will reveal a systolic or presystolic murmur and a mid-diastolic murmur, in the mitral area, in addition to the early diastolic murmur of pulmonary incompetence (Fig 120) This combination of auscultatory signs causes a difficulty in clinical diagnosis of mitral stenosis combined with aortic incompetence

from Lutembacher's syndrome. The former condition is likely if the pulse is large, whereas if the early diastolic murmur cannot be heard to the right of the mid-line the second condition is suggested.

Confirmation of the diagnosis comes from *cardioscopy*, which will show the characteristic changes of auricular septal defect when enlargement of the right



Fig 121.—Auricular septal defect and mitral stenosis (Lutembacher's syndrome) (A) Anterior view (B) Right oblique view Enlargement of the pulmonary artery (1) and its branches (2) which showed great pulsation on cardioscopy (hilar dance) Great enlargement of right auricle (3) Elongated left bronchus impression (4) from distension of the right pulmonary artery. Prominent left auricular impression (5) Left ventricle (6) displaced to the left by the big right heart

side of the heart is particularly prominent, and when distension of the left auricle shows in the right oblique position with barium in the oesophagus (Fig. 121)

VENTRICULAR SEPTAL DEFECT

Although a defect of the ventricular septum may often produce a conspicuous murmur, it is not a great mechanical hindrance to the heart unless the defect is a large one or associated with other congenital deformities Its serious implication arises from the attendant liability to bacterial endocarditis The opening is usually situated towards the base and just anterior to the membranous septum, so that the bundle of His which courses behind is often undisturbed. Some 4 out of 5 cases of ventricular septal defect show other congenital abnormalities among which those making up Fallot's syndrome are the commonest. When it exists as the only lesion the opening is usually small, although rarely it may be large enough to constitute a trilocular heart.

SYMPTOMS AND DIAGNOSIS

In that the shunt in ventricular septal defect is of the arterio-venous type, cyanosis is uncommon. It is rare for pulmonary disease or heart failure to reverse the

shunt, so that if cyanosis is present it means that some other congenital anomaly, particularly pulmonary stenosis, is associated with the septal defect. Again,

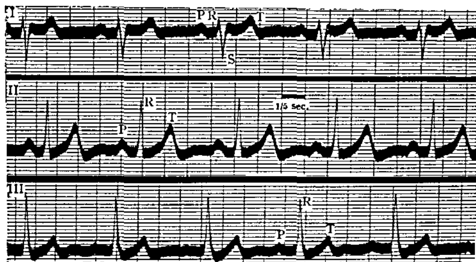


FIG. 122—Ventricular septal defect showing right axis deviation

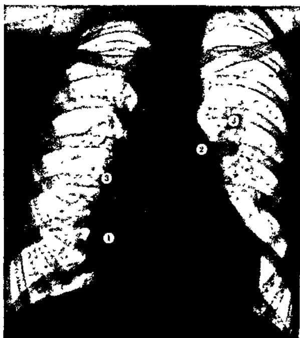


FIG 123—Ventricular septal defect. Enlargement of the right auricle (1), pulmonary artery (2), and its branches (3)

owing to the infrequency of cyanosis and heart failure, breathlessness is an unusual symptom. A rough systolic murmur is localized in the fourth intercostal space

near the left border of the sternum and is accompanied by a thrill. A systolic murmur in this position without a thrill usually means that the murmur is of the innocent kind (Figs. 33, 34 and 35).

The *electrocardiogram* is often physiological, or it may show right electrical axis deviation (Fig. 122).

On *cardioscopy* the heart shows some enlargement of the right auricle, the right ventricle, and the pulmonary artery (Fig. 123).

PROGNOSIS

The condition may not give rise to any symptoms and may be discovered fortuitously during routine examination Yet, some one-fourth of the cases develop bacterial endocarditis when vegetations may form either at the edge of the opening or sometimes on the mural endocardium of the right ventricle opposite the opening In a series of 50 cases the average age at death was 14 years and the maximal age 49 The influence which ventricular septal defect has on a case of pulmonary stenosis is fourfold ; the thrill in the pulmonary area is usually short or absent , the systolic murmur, loudest in the pulmonary area, is often conducted to the aortic area and into the vessels of the neck ; if the ventricular septal defect is large and the pulmonary artery rudimentary, there may be no murmur in the pulmonary area , a ventricular septal defect improves the prognosis in pulmonary atresia, but makes it less favourable in pulmonary stenosis.

PATENT DUCTUS ARTERIOSUS

The ductus arteriosus may remain patent in the absence of any other congenital abnormality of the heart. It occurs commonly in association with other developmental anomalies, and especially when they are of the severe type. Its diameter, usually greatest at the aortic end, is variable and may only admit a probe ; if it is large enough to admit a pencil it might undergo aneurysmal dilatation. The pulmonary artery becomes dilated and the right ventricle and auricle become enlarged, as also does the left ventricular sometimes. The extent of these changes depends on the size of the lumen of the ductus.

SYMPTOMS AND DIAGNOSIS

Cyanosis and breathlessness are absent in patent ductus arteriosus as long as the shunt remains arterio-venous in type. In the presence of other congenital defects, terminal heart failure, pulmonary disease, or exertion, the shunt may reverse into the veno-arterial type when cyanosis and breathlessness may appear

With a moderately large ductus the pulse may be collapsing in character with a raised pulse pressure , this effect is sometimes made more obvious by exercise, but this is by no means always so The apex beat may be displaced to the left if there is much enlargement of the right heart. A systolic thrill is felt in the pulmonary area in more than one-half the cases. In the same area a rough systolic murmur is heard ; it is long and lasts into diastole. For this reason it has been described variously as like a humming top, a rolling sea, machinery, or as a continuous murmur. The second sound in the pulmonary area is usually loud within the murmur, which at this phase is intensified (Figs. 124 and 125).

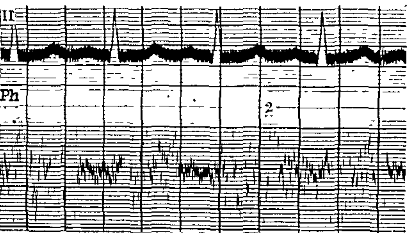


FIG 124.—Patent ductus arteriosus The phonocardiogram shows a continuous murmur lasting throughout systole and diastole with intensification in early diastole (2)

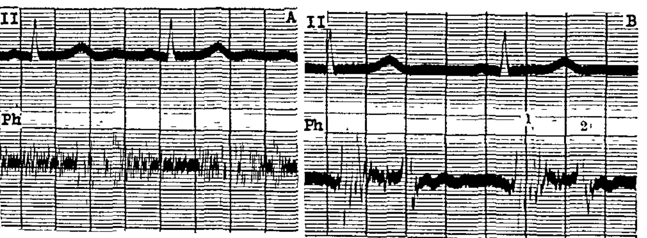


FIG 125.—Patent ductus arteriosus The continuous murmur in (A) has disappeared in (B) which was recorded after ligation of the duct

The *electrocardiogram* is normal, so that it is an important test in cases of doubt, for should the cardiogram prove abnormal a diagnosis other than patent ductus arteriosus has to be entertained.

The changes found on *cardioscopy* depend on the size of the ductus, so that if the lumen is small there may not be any alteration in the cardiac silhouette or at the most only slight enlargement of the pulmonary arc. If the ductus is widely

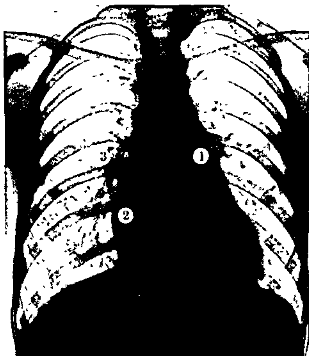


Fig 126—Patent ductus arteriosus in a child aged 9 years Enlargement of the pulmonary artery (1), and fulness of the right auricle (2) Prominence of the hilar markings (3)

patent the pulmonary artery and its branches are prominent, and there is moderate enlargement of the heart, especially of the left ventricle and of the right heart (Fig 126).

PROGNOSIS

In a series of 92 cases the average age was 24 and the maximum 66 years. Many patients remain unhandicapped by the lesion, although the outlook must always remain uncertain in the absence of surgical treatment in that some one-fifth of the cases develop bacterial endocarditis. In this event the vegetations are usually found at the pulmonary artery extremity of the ductus. In the uncomplicated cases the prognosis depends on the size of the opening and the extent of cardiac enlargement.

TREATMENT

In view of the high incidence of bacterial endocarditis and ultimate enlargement and failure of the heart, ligation of the ductus may become routine practice at an early age. The incision should be at the back.

COARCTATION OF THE AORTA (OPEN DUCTUS)

The presence of a patent ductus, situated proximal to the aortic constriction, produces a different anatomical and clinical picture from that presented by coarctation with a closed ductus. Thus, with an open ductus the right heart may be enlarged while the left ventricle may be unaltered. The aorta proximal to the site of the stenosis is not dilated and hypertrophied arteries forming a collateral anastomosis are not a feature.

The signs are similar to those found in patent ductus arteriosus, but additionally it may be noticed that the amplitude of the femoral pulse is very small, whereas it is normal or even increased in a case presenting a patent ductus arteriosus without aortic coarctation The systemic blood pressure is not raised.

The outlook here is much less favourable than in coarctation with a closed ductus, and not many patients survive early childhood.

GROUP III WITH CYANOSIS (Veno-arterial shunt)

PULMONARY STENOSIS (COMPLICATED)

Three anatomical types of pulmonary stenosis may be recognized according to the site of the narrowing In each there is enlargement of the right auricle and right ventricle, but the state of the pulmonary artery varies and this factor assumes importance in the radiological diagnosis of the condition. In about three-fourths of the cases a defect of the ventricular septum is also present and this group is discussed later under Fallot's syndrome.

Conus stenosis.—In this type the right ventricle is divided into two compartments, the upper one, which includes the conus, is dilated and so is the pulmonary artery as far as its bifurcation or beyond In most cases there is an opening in the ventricular septum

Valvular stenosis.—In this variety the pulmonary cusps are thickened and are fused together leaving a central aperture The pulmonary artery beyond is dilated There may be no other congenital abnormality, although ventricular septal defect is sometimes present

Arterial stenosis —Here the narrowing is confined to the pulmonary artery beyond the cusps, although these may occasionally be thickened The pulmonary artery is always small, and a ventricular septal defect is present in some three-fourths of the cases

SYMPTOMS AND DIAGNOSIS

In the *lone* lesion, cyanosis may appear late due to heart failure and it is never prominent. Inasmuch as a defect of the ventricular septum so commonly accompanies pulmonary stenosis, it follows that most cases will demonstrate cyanosis, breathlessness and arthropathy; this is why pulmonary stenosis is included in the cyanotic group, but it needs emphasis that the lone lesion seldom produces cyanosis. The pulse is rather small as a rule. The apex beat may be displaced outwards because of enlargement of the right side of the

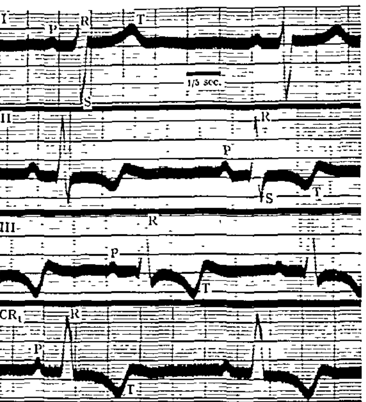


FIG 127.—Pulmonary stenosis Right axis deviation, and inversion of the T wave in leads II, III and CR_1 No S wave in CR_1 The T wave was upright in CR_7.

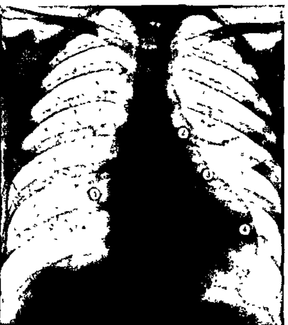


FIG. 128.—Pulmonary stenosis Enlargement of the right auricle (1), pulmonary artery (2) and conus (3). The left ventricle (4) was enlarged because of associated hypertension There is no hilar congestion. (Post-mortem control.)

heart, which may produce increased pulsation in the epigastrium. A systolic thrill is usually present over the pulmonary area A rough systolic murmur is heard in the same area and the intensity of the second sound is variable; in valvular and arterial stenosis the pulmonary second sound is usually absent, but in conus stenosis it is often accentuated. Occasionally an early diastolic murmur is present. It is of great clinical importance to tell the innocent systolic murmur in the pulmonary area from pulmonary stenosis. The former is not so loud, becomes less loud in the upright posture and on deep inspiration, is never accompanied by a thrill, and is situated in mid-systole.

The *electrocardiogram* shows right electrical axis deviation and inversion of the T wave in leads II, III and CR_1 (Fig. 127), but sometimes it shows only minor changes such as inversion of T in lead III.

Cardioscopy shows only slight enlargement of the right auricle and right ventricle. According to the type of stenosis the pulmonary arc is either absent (two-thirds of the cases) or prominent (one-third of the cases). Pulmonary congestion is minimal as a rule, even in heart failure (Fig. 128).

PROGNOSIS

Disability is not noticed in lone pulmonary stenosis until adult life, because cyanosis and breathlessness are late features of the condition. In a series of 9 cases which showed no associated congenital lesion the average age was 22 and the maximal age 45 years. Pulmonary stenosis is subject to the same complications as are other congenital heart lesions, namely, bacterial endocarditis, heart failure, and less commonly pulmonary infection.

PULMONARY ATRESIA (COMPLICATED)

The pulmonary artery in pulmonary atresia either is a fibrous cord or is canalized for a distance ending in a cul-de-sac Two types are recognized.

PULMONARY ATRESIA WITHOUT VENTRICULAR SEPTAL DEFECT

Here the lesion is usually associated with a defect of the auricular septum and a patent ductus arteriosus. Cyanosis is extreme from birth, and as patients with this condition do not survive long, the syndrome has no clinical importance.

PULMONARY ATRESIA WITH VENTRICULAR SEPTAL DEFECT

Patients in this group frequently survive to adult life and account for three-fourths of all cases of pulmonary atresia, and one-fifth of all cases of Fallot's syndrome. *A special anatomical feature of this group is the presence of hypertrophied* bronchial arteries forming a collateral circulation. Enlargement of the right auricle and right ventricle is present and the pulmonary artery is small. The ductus arteriosus may also be present.

SYMPTOMS AND DIAGNOSIS

The clinical features of pulmonary atresia will be dealt with under Fallot's syndrome, and only two of its special characteristics are mentioned here. First, cyanosis is very conspicuous from birth and arthropathy develops early. Secondly, a murmur is not heard, as a rule, in the pulmonary area and when a murmur is audible it arises from the associated ventricular septal defect.

FALLOT'S SYNDROME

The association of pulmonary stenosis or atresia with a defect of the ventricular septum, and a biventricular aorta (aortic opening astride the septal defect), constitutes Fallot's syndrome. Enlargement of the right side of the heart is a *natural sequence. Pulmonary stenosis is present in some four-fifths of the cases* and pulmonary atresia in one-fifth A right-sided aortic arch is sometimes present This group of cases assumes importance in the fact that some three-fourths of all cases of congenital heart disease which show cyanosis are instances of Fallot's syndrome; the proportion is even greater if the analysis is confined to adults.

SYMPTOMS AND DIAGNOSIS

Cyanosis is prominent from the start; it progresses and often becomes extreme Arthropathy develops in association with the cyanosis. Similarly, dyspnoea

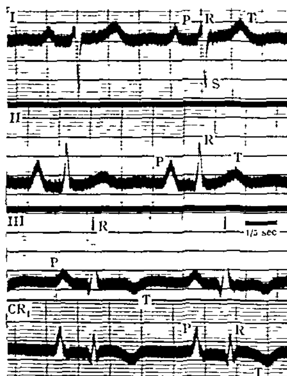


FIG 129 —Fallot's syndrome. Right axis deviation. Inversion of T wave in leads III and CR_1 The P wave is tall in II and CR_1 The S wave in CR_1 is small

on exertion is a conspicuous symptom, and is commensurate with the degree of cyanosis In infants, attacks of unconsciousness and convulsions occur and death during such attacks is not uncommon The pulse is usually small, but arterial pulsation is often noticeable in the neck. The apex beat is displaced outwards to the left and is proportionate to the degree of enlargement of the right side of the heart, which may be slight or which may produce prominent

pulsation in the epigastrium. A systolic thrill in the pulmonary area is less common than in cases of lone pulmonary stenosis and it is absent in atresia. A rough systolic murmur is expected in the pulmonary area, but it is modified by two circumstances; if the opening in the ventricular septum is large the murmur is less loud and is conducted into the aortic area, and if pulmonary atresia is present there is no pulmonary murmur. The second sound in the pulmonary area is commonly distant or even absent, but sometimes it is loud.

The *electrocardiogram* (Fig 129) usually shows right electrical axis deviation, and often inversion of T waves in II, III and CR_1 as well, so that if a patient with cyanosis from congenital heart disease does not have this characteristic tracing, the diagnosis of Fallot's syndrome should be in doubt

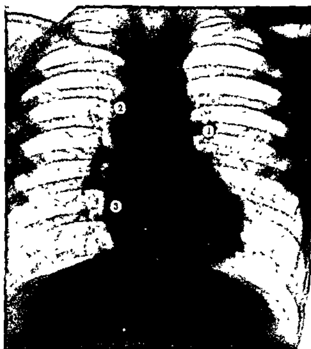


FIG. 130 —Fallot's syndrome The pulmonary bay (1) is empty The aortic shadow is prominent to the right (2) Fulness of the right auricle (3)

The findings at *cardioscopy* vary according to the origin and distribution of the aorta. Thus, there are three types In the *first* type, which is the commonest, the aorta is normal at its origin and in its course; enlargement of the right auricle, right ventricle, and pulmonary artery is slight or moderate, so that the normal cardiovascular contour is fairly well preserved and resembles that found when either pulmonary stenosis or ventricular septal defect occurs alone. In the *second* type (Fig. 130) the aorta has a biventricular origin arising astride the septal defect, so that the ascending portion of the aorta is prominent on the right; the pulmonary bay is empty and there is a prominence of the toe of the left ventricle from enlargement of the right heart. In the *third* type (described below) the pulmonary lesion and septal defect are associated with a right-sided aortic arch.

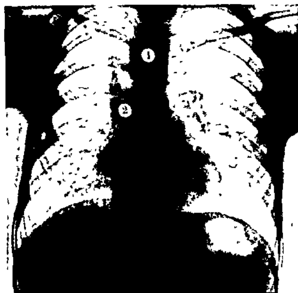


FIG 131 —Right-sided aortic arch. The aortic arch impression on the oesophagus (1) is reversed. The aortic knuckle is absent, and the descending aorta (2) is seen on the right Child aged 7 years with Fallot's syndrome



FIG 132 —Right-sided aortic arch The aortic arch impression (1) in the right oblique position is reversed. Child aged 15 years with Fallot's syndrome.

PROGNOSIS

In a series of 74 cases the average age was 12 years and the maximal 59 years. The outlook is less satisfactory when pulmonary atresia is present, although some of the patients survive to adult life. Bacterial endocarditis is less common in Fallot's syndrome than in many other examples of congenital cardiac defects Heart failure and pulmonary infection are common, and cerebral accidents occur.

TREATMENT

The alleviation of cyanosis in these cases by transplanting the subclavian into the pulmonary artery appears sometimes worth while, but its true value has yet to be tested.

EISENMENGER'S SYNDROME

This condition is uncommon and consists of the following anatomical deformities—dilatation of the pulmonary artery, ventricular septal defect which is usually large, and biventricular aorta The right side of the heart is enlarged.

Cyanosis may not be prominent and may appear late. In the same way breathlessness and arthropathy are late features A systolic murmur may be heard just below the pulmonary area and a diastolic murmur from pulmonary incompetence is audible

On cardioscopy the right auricle and conus of the right ventricle are seen to be enlarged, and there is prominence of the pulmonary arc and the hilar shadows, representing the branches of the pulmonary artery, which may also show excessive pulsation

RIGHT-SIDED AORTIC ARCH

In this condition the aorta takes a right-sided curve and arches over the root of the right lung, passing backwards to the right of the trachea and oesophagus. It then descends on the right side of the spine before it crosses to the left to gain the aortic opening in the diaphragm Sometimes the aorta turns abruptly to the left after crossing the right bronchus, and widens into an ampulla which displaces the trachea forwards and to the left. The left subclavian artery arises from this ampulla and passes to the left behind the trachea and oesophagus. Sometimes the left subclavian arises from the ascending aorta and crosses to the left in front of the trachea and oesophagus. The condition results from a suppression of part of the fourth embryonic arch on the left side and its persistence on the right side. It may appear as a sole congenital anomaly, but more often it is found in association with Fallot's syndrome.

It has no influence on prognosis and its recognition is only possible on *cardioscopy* (Figs 131 and 132). The aortic knuckle may appear small or its place may be taken by a shadow representing the ampullary projection of the arch. The descending aorta is seen on the right. With barium in the oesophagus the aortic arch impression is seen to be reversed with its concavity directed to the right instead of to the left. Displacement of the trachea to the left is also seen at this level. The features of Fallot's syndrome may also be present.

CHAPTER 6

DISEASES OF THE PERICARDIUM

AETIOLOGY AND PATHOLOGICAL VARIETIES

ACUTE rheumatism, pneumonia, tuberculosis, and certain of the infectious fevers are amongst the commoner causes of pericarditis in young subjects. In older patients it is sometimes a complication of chronic nephritis and of the terminal stages of other serious illnesses, and in a more localizing form it is found associated with cardiac infarction Purulent pericarditis may result from a general systemic pyaemia due to staphylococcal, streptococcal, or pneumococcal infection. It may also follow a direct spread from an adjoining focus of suppuration as in empyema, or it may be the result of trauma Haemorrhagic pericarditis, in which the exudate is stained red, is the outcome of tuberculosis or malignant disease, and should be distinguished from haemopericardium, which results from rupture of the heart or the first part of the aorta

Most affections of the pericardium involve the whole of the pericardial sac and its covering, and the symptoms which they produce are the outcome of some or all of the following effects: constriction of the heart, pressure on adjoining structures, local injury to the myocardium, and toxaemia In the early stages of acute fibrinous or dry pericarditis, the exuded lymph coagulates on the smooth surface of the pericardium causing it to lose its lustre. This layer of fibrin may gain in thickness and assume a ragged honeycombed pattern ("bread and butter" pericarditis). When healing has taken place the pericardium may be slightly thickened or the resulting adhesions may bind together the visceral and parietal layers and obliterate the sac. Should the inflammatory process spread outside the pericardium, and especially when it is tuberculous in nature, the adhesions may anchor the heart to adjacent structures, notably the mediastinum, pleura and diaphragm. In the constrictive type there is great thickening of the pericardium and calcification is common; within its layers there is sometimes an accumulation of creamy material. This chronic inflammatory process extends for a short distance into the myocardium and for this reason produces changes in the electrocardiogram. Serofibrinous pericarditis commences as the fibrinous form and is followed by a serous exudation which may fill and even distend the sac.

SYMPTOMS AND SIGNS

The signs traditionally attributed to pericardial disease need to be revised for three reasons First, a correlation of the clinical and pathological findings in cases judged to have adherent pericardium has shown how unreliable the physical signs have been. Secondly, the signs comprising Pick's syndrome associated with constrictive pericarditis have frequently appeared in other types of pericardial disease Thirdly, the help gained by cardioscopy and electrocardiography has demanded for these methods a more prominent place in the diagnosis of pericardial disease. It is, therefore, expedient to discuss the signs assigned to the condition under three headings which help to emphasize the common presenting signs and to forbear emphasizing those which are less common and especially those of doubtful accuracy.

Equivocal signs

The majority of these signs have been assembled in the past to support a diagnosis of adherent pericardium, and less frequently, of effusion, but their correlation with findings of cardioscopy and necropsy has proved them to be unreliable as evidence of pericardial disease. Thus, *undulatory pulsation* from the base to the apex of the heart is often seen in healthy thin subjects, or in thin patients with enlargement of the heart from any cause Again, *peri-apical systolic retraction* is commonly met with under the same circumstances. Systolic retraction of intercostal spaces of the lateral and posterior walls of the left chest (*Broadbent's sign*) is by no means a sure sign of pericardial disease, nor is diastolic collapse of the veins of the neck (*Friedreich's sign*) Lateral instead of antero-posterior expansion of the chest during inspiration (*Wenckebach's sign*) is hardly ever seen in pericardial disease A *presystolic murmur* has been described in adherent pericardium, but should this auscultatory sign be found it should lead to a diagnosis of mitral stenosis Adherent pericardium does not in itself give rise to *cardiac enlargement*, thus in one-half of a series of cases with adherent pericardium examined at necropsy the heart was not enlarged ; in the other half, cardiac enlargement had always resulted from some other cause, usually valvular disease, and was proportionate to the severity of such a lesion In the case of pericardial effusion it is stated that dulness to percussion may be elicited at the cardiohepatic angle (*Rotch's sign*), but, in my experience, radiology of pericardial disease has never shown an obliteration of this angle by pericardial effusion.

Circumstantial signs

Included in this group of symptoms and signs are those which may arise in individual patients and are the outcome of circumstances which result in the course of the disease or as a complication of it.

Thus, at the onset of acute pericarditis *pyrexia* is common ; if it continues it may mean that the pericardial effusion shows suppuration or that its nature is tuberculous. In the course of a general systemic infection the onset of pericardial involvement is sometimes signalled by a rigor. *Pain* in the chest sometimes accompanies a rapidly forming pericardial effusion, but is seldom prominent in acute pericarditis. It is naturally present when cardiac infarction is a cause *Fremitus* and a to-and-fro *friction sound* are commonly present in acute pericarditis. The characteristic sound sometimes resembles closely the systolic and diastolic murmurs of aortic incompetence. *Pallor*, *dyspnoea* and *restlessness* usually express the toxic effects of the illness as much as the results of the pericardial disease itself. When the effusion is large, *dysphagia*, *distressing cough* and *hiccough* may set in because of the pressure on the oesophagus and phrenic nerve. Dulness of the left pulmonary base, tubular breathing, and aegophony (*Bamberger's sign*), may follow the pressure of a pericardial effusion on the lung ; it is seldom the result of pneumonic consolidation. *Albuminuria* is present when pericardial effusion has formed in nephritis. *Adenopathy* in the neck or elsewhere may help to show that tuberculosis or some other granuloma has caused the pericardial lesion, or rarely, a neoplasm. An associated tuberculous *arthritis* will also assist in the diagnosis of tuberculous pericarditis and so will the presence of *pulmonary tuberculosis*. If the pericardial effusion is *haemorrhagic* it is likely to be the outcome of tuberculosis, neoplasia or, more rarely, rupture of the heart or aorta.

Characteristic signs

The signs now to be described, conveniently grouped under three headings, are common to all types of pericardial disease, but if the pericardial condition resolves, the signs are transitory and quickly disappear even before they are well formed. Should the signs last and become prominent they confirm the presence of *constrictive pericarditis.*

Signs common to right heart failure.—The signs similar to those found in failure of the right heart from any cause include cyanosis, distended cervical veins, distended liver, ascites, oedema of the ankles, small pulse sometimes with

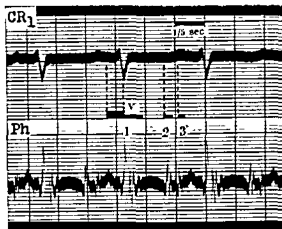


FIG 133.—Constrictive pericarditis. Triple heart rhythm from the addition of the third heart sound in a female patient aged 63 years

auricular fibrillation, low blood pressure, and triple heart rhythm from addition of the third heart sound When pericarditis is the cause the oedema is less prominent than in right heart failure and the ascites more prominent as a rule; distension of the liver, too, is much greater The third heart sound is loud and clear in the established case of constrictive pericarditis and is a sign of great importance in clinical diagnosis (Fig. 133)

Signs dissimilar from those in right heart failure—Breathlessness is not a common presenting symptom of pericarditis and it is never present at rest Again, pulmonary congestion is not prominent so that basal crepitations are absent; if they are present they are related to the local abnormal lung condition. Moreover, unlike other examples of right heart failure, pericarditis does not cause enlargement of the heart, and murmurs are absent.

Confirmatory signs—Confirmation of the diagnosis of pericarditis comes from examination by cardiography and cardioscopy.

The electrocardiogram

The changes in the cardiogram of *acute pericarditis* are confined in the early stages to the R-T segment. Like those in cardiac infarction they are short-lived and

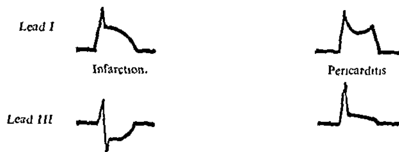


FIG 134 — Design of the electrocardiogram in leads I and III in early cardiac infarction and in acute pericarditis

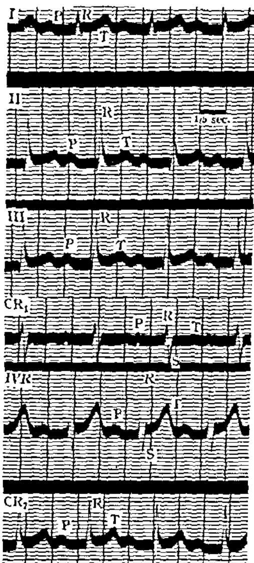


FIG. 135 — Acute pericarditis Elevation of the R–T segment with upright T wave producing a saddle curve in leads II, III, IVR and CR_7

are followed by inversion of the T wave. The form of the R–T deviation is different from cardiac infarction, and so is its direction in leads I and III Thus, the elevated R–T of anterior cardiac infarction in lead I shows coving and the convexity of the curve is upwards, whereas the elevated R–T of pericardial disease shows an upward concavity or a saddle curve. Again, the reciprocal relationship between leads I and III in cardiac infarction, namely, elevation of the R–T segment in one lead and depression in another, is absent in pericarditis (Figs 134 and 135) Although the duration of the R–T changes is no longer than a few days,

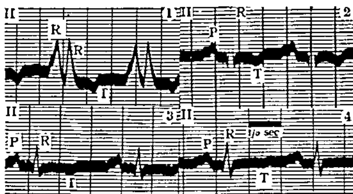


FIG 136—Shelving effect in P_2 in four patients with constrictive pericarditis

the deformity of the T wave which follows will last or disappear according to the progress of the underlying myocardial injury, and it does not depend on the presence or absence of pericardial fluid

The cardiogram of *constrictive pericarditis* shows lasting changes which are not altered by time or even by a successful operation. In such a tracing the voltage is sometimes low ; the rhythm is often irregular from auricular fibrillation even in young patients ; the P wave is usually broad and often demonstrates a shelving effect in lead II (Fig. 136) ; the Q wave is never prominent, and the R–T segment seldom shows any deviation Changes in the T wave make it convenient to consider them in two groups, although all cases do not conform to this grouping, and occasionally, in patients in whom the adherence of the pericardium to the heart is not severe, the T waves may be upright.

In the *first type* (Fig. 137) the T wave is inverted in leads III, II, CR_4, and in CR_1, but it is upright in I and in CR_7

In the *second type* (Fig 138) the T wave is inverted in leads I, II, CR_4, and in CR_7. The T wave in CR_1 is either low or inverted.

Cardioscopy

On cardioscopy in *pericardial effusion* the cardiac silhouette is enlarged both to the right and to the left ; the right border is displaced outwards in the region of the right auricle and for some way above it, and on the left side there is extension outwards at the left ventricle, while the pulmonary bay above is filled in producing a straighter left border as high as the aortic knuckle, so that the vascular stem

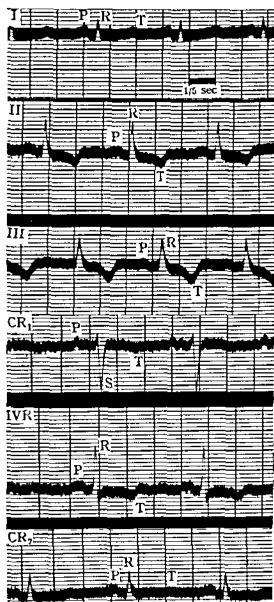


FIG 137 —Constrictive pericarditis The T wave is inverted in leads III, II, IVR and in CR_1 it is upright in I and CR_7.

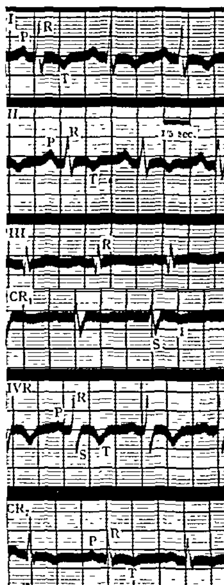


FIG 138 —Constrictive pericarditis The T wave is inverted in leads I, II, IVR, CR_7 and CR_1



Fig. 139.—Pericardial effusion (A) taken before treatment and (B) after partial paracentesis and air replacement; the exposed upper part of the heart shadow shows that there is not any cardiomegaly. (C) was taken six weeks later after complete absorption of the fluid

is shortened. The outward and upward extension of the normal cardiac silhouette by distension of the pericardium is well demonstrated by replacing some of the effusion by air (Fig 139). Owing to the diminished pulsation of the pericardium a teleradiogram will show a stencilling effect (Fig 140). In the right oblique position the left auricular impression is prominent and the barium stream may be separated from the dense heart shadow by a lighter area When there is no heart disease present, a second teleradiogram after the absorption of the pericardial effusion shows a more transparent as well as a smaller heart shadow (Fig 141). In the presence of heart disease the heart shadow is less after the absorption of fluid, but it remains abnormal according to the effects of the underlying disease (Fig. 142)

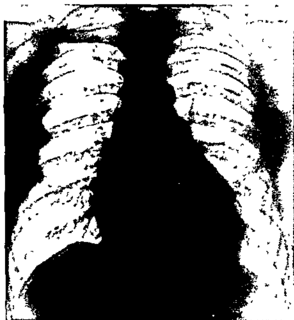


Fig 140—Pericardial effusion Stencilled outline of the heart border in a male aged 56 with heart failure after cardiac infarction

On cardioscopy in *constrictive pericarditis* the cardiac silhouette appears slightly enlarged, and shows little movement, so that the teleradiogram shows a stencilled appearance. In the oblique positions the left auricular impression on the oesophagus is increased , this appearance is like that seen in mitral stenosis, but when the effect is produced by pericardial disease a light area is usually seen between the denser shadow of the heart and the barium stream, whereas in mitral stenosis no such light area is discernible and the dense shadow of the left auricle is contiguous with the barium stream (Fig. 143) If calcification has taken place (Figs 144, 145 and 146) the diagnosis of constrictive pericarditis is confirmed, provided that the clinical signs are also present, but this may be missed occasionally if radiological examination is confined to cardioscopy and does not include a teleradiogram

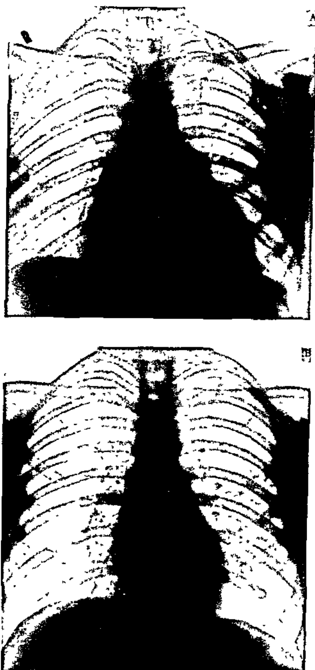


FIG 141 —Pericardial effusion in (A) is absent in (B) taken a month later The effect of the effusion has been to enlarge the cardiac shadow and give it a stencilled appearance The vascular stem is shortened and the natural hilar markings are hidden.



FIG 142—Pericardial effusion from heart failure in mitral stenosis The straight stencilled left border before treatment (A) has given way to four arcs in (B) taken after treatment. Aortic knuckle (1) Pulmonary arc (2) Pulmonary conus (3), Left ventricle (4)



Fig 143 —Constrictive pericarditis. In the right oblique position a light area at the left auricular impression (1) separates the barium stream from the denser heart shadow Arrows indicate calcified areas Male aged 28 years.



Fig 144 —Constrictive pericarditis. Calcification (indicated by arrows) is widespread, and is best seen at the left border in the anterior view



FIG 145—Constrictive pericarditis In the left oblique position a light area at the left auricular impression (1) separates the barium stream from the denser heart shadow Arrows indicate calcified areas Male aged 28 years



FIG 146—Constrictive pericarditis in right oblique position. Arrows indicate calcified areas Left auricular impression (1) Male aged 47 years

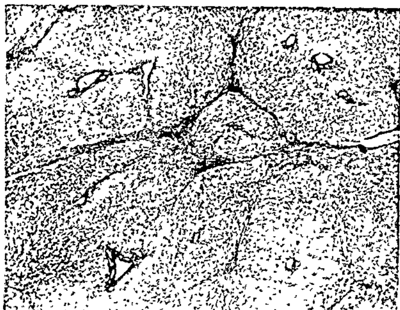


FIG 147 —Early congestion cirrhosis in a male aged 17 years whose symptoms of constrictive pericarditis disappeared with the operation of cardiac decompression Death 8 months later was from tuberculous meningitis. (Azan stain. ×22)



FIG. 148 —Severe congestion cirrhosis in a female aged 51 years whose symptoms of constrictive pericarditis, notably ascites, persisted after cardiac decompression till her death 22 months later. (Azan stain ×22)

DIFFERENTIAL DIAGNOSIS

When the clinical signs have been assembled, when examination of the urine has excluded nephritis as a cause, and when absence of heart murmurs has ruled out heart failure from valvular disease, there remains one condition, namely pulmonary hypertension, which closely resembles the clinical picture presented by pericardial effusion To distinguish the conditions it is a help to remember that pulmonary hypertension does not affect young subjects and that certain differences may show during electrocardiographic and cardiographic examinations. Thus, the cardiogram in pulmonary hypertension will show deviation of the electrical axis to the right as well as inversion of the T wave in leads II, III and CR_1. On cardioscopy in pulmonary hypertension it will usually be possible to find enlargement of the pulmonary artery in addition to some pericardial effusion which is commonly present as part of the accompanying heart failure.

COURSE AND PROGNOSIS

In acute pericarditis the immediate prognosis in the majority of cases is favourable, but distress, restlessness, and even delirium are more ominous signs which may indicate the presence of acute endocarditis in addition to pericarditis. When recovery from uncomplicated acute pericarditis takes place the ultimate prognosis is excellent and usually the patient is none the worse for the illness. Even adherence of the parietal and visceral layers of the pericardium which has obliterated the sac is not an uncommon finding at necropsy in patients who have died from some other disease and who did not show any symptoms during life Naturally the prognosis is serious when pericarditis is a complication of nephritis, pulmonary tuberculosis, pyaemia and pneumonia, or when the effusion is purulent or haemorrhagic. In the insidious form of the disease (constrictive pericarditis) with symptoms caused by cardiac compression, the patient's condition gradually deteriorates in the course of a few years unless it is relieved by operation In a series of cases coming to operation I have found that the ultimate success attendant on the surgical procedure of cardiac compression depends on the state of the liver at the time. Thus, if the condition has prevailed for a long time, congestion cirrhosis (Figs 147 and 148) might be so great as to obscure the beneficial effect gained from the operation. On the other hand, if surgical treatment is carried out early and before the changes in the liver have become gross, the improvement may be so noticeable that mercurial diuretics can be discontinued immediately or soon after the operation (Fig. 149)

TREATMENT

In acute pericarditis treatment needs to be directed to the general condition causing it, and aimed at relieving symptoms incidental to it. When pericardial effusion, which is usually tuberculous and seldom rheumatic, is forming rapidly, paracentesis should be undertaken along with air replacement, for it may ease the pain and distress, and help the process of absorption The site of the puncture is either in the fifth left intercostal space in the mammary line or in the angle between the ensiform cartilage and the lower sternal border, when the exploring needle should be directed upwards behind the sternum If suppuration is present repeated aspiration or drainage of the pericardium should be carried out, and the last site of puncture should be used in order to avoid spreading the infection to the pleura.

In the insidious or constrictive type of pericarditis, with or without calcification, it is necessary at first to watch the patient during a period of rest and treatment with mercurial diuretics As soon as it becomes clear that the symptoms of ascites and oedema are not benefited by rest alone, and that on a standard fluid intake of 30 ounces daily the output is consistently lowered (usually less than 20), and that fluid retention can only be prevented by the continued use of mercurial diuretics, partial resection of the pericardium should be carried out by an experienced thoracic surgeon. Naturally this operation is a serious undertaking and carries with it inevitable risks. Even when the immediate hazards of the operation

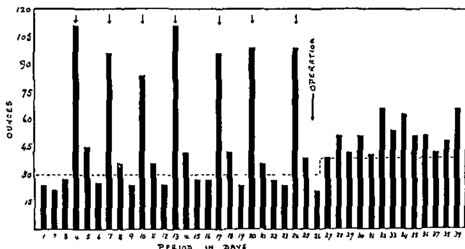


FIG 149 —Fluid intake (discontinuous line) and urinary output (black columns) in a man aged 43 years with constrictive pericarditis, before and after cardiac decompression Arrows indicate intravenous injections of Neptal (2 c c)

have been passed the symptoms are not relieved in all cases, and complications such as generalized dissemination of the tuberculous infection may take place On the other hand, the operation in many patients has produced great improvement after a period of a few months during which the use of mercurial diuretics needs to be continued. The aim of the surgeon should be to remove as much as possible of both layers of the pericardium from the anterior surface of the right ventricle and from the left and posterior parts of the left ventricle Clearing of the auricles is a hazardous procedure and should not be undertaken because it is unnecessary. Relief takes place as the result of cardiac decompression because herniation of the heart through the opening in the pericardium is a noticeable feature at operation and afterwards when examined by cardioscopy. It has already been pointed out that the pace of the improvement after successful cardiac release is mostly dependent on the extent of congestion cirrhosis present and to a much lesser extent on the advent of such complications as miliary tuberculosis It bears repetition, therefore, that the diagnosis of constrictive pericarditis should be made early so that the operation of cardiac decompression may be carried out before congestion cirrhosis has advanced so far as to outweigh the benefits of a successful operation.

CHAPTER 7

ENDOCARDITIS

THERE is no simple orthodox way of discussing the subject of endocarditis. Sometimes the *course* of the illness receives emphasis and it is described as acute, subacute, or chronic. The character of the *vegetations* necessitates a division into the two main groups of granulomatous, or verrucose, and septic, or ulcerative The *severity* of the condition is given emphasis when it is described under the headings of simple and malignant. Again, its *anatomical* distribution is specially taken into account when endocarditis is discussed in relation to the valves which are affected.

It is intended here to describe endocarditis in relation to its *aetiology*. Thus we have pneumococcal, gonococcal, streptococcal and rheumatic endocarditis. Staphylococcal endocarditis as part of a pyaemia complicating otitis media, osteomyelitis, and puerperal fever, has no special clinical significance. Similarly, tuberculous endocarditis, usually developing in the course of disseminated miliary tuberculosis, is largely of pathological interest. Syphilitic aortic endocarditis results from the extension of aortitis and will be considered specially under that heading.

PNEUMOCOCCAL ENDOCARDITIS

Pneumococcal endocarditis is usually, although not invariably, associated with pneumonia, and it is present in about 10 per cent of all cases of pneumonia coming to necropsy; among these, empyema is a common complication The left side of the heart is more susceptible to the infection than the right, and the aortic valve is particularly vulnerable. Previous damage to the valve has a predisposing effect. The yellowish-green vegetations which form are usually large and sometimes even massive, depending on the length of the illness. They are single, as a rule, and tend to be polypoid, showing a characteristic smooth surface without ulceration.

SYMPTOMS AND SIGNS

The symptoms of septicaemia are uppermost, but embolic effects may appear as well as heart murmurs. In the presence of pneumococcal endocarditis other complications of pneumonia, particularly meningitis, are common Pericarditis developing during or after the pneumonia should always lead to the suspicion that the endocardium has also become infected A positive blood culture will establish the diagnosis.

TREATMENT

Prevention should be the chief aim of treatment, for when pneumococcal endocarditis has developed, sulphonamides, sera and penicillin are all without effect on the general course of the illness.

GONOCOCCAL ENDOCARDITIS

Gonococcal endocarditis occurs in the course of gonococcal bacteriaemia or pyaemia. The vegetations are situated more commonly on the aortic than the mitral valve, which may or may not be thickened by previous disease

SYMPTOMS AND SIGNS

The true relationship of the symptoms to the onset of the primary infection is inconstant, but as a rule it is a matter of weeks or sometimes months in the male. In the female the absence of a cervical discharge and even negative bacteriological examination of cervical and urethral smears does not exclude the diagnosis.

The symptoms for the most part are related to the septicaemia, but embolic phenomena may be observed, and cardiac murmurs may appear if a valvular lesion already exists A blood count shows leucocytosis with a relatively greater increase in the polymorphonuclear cells. Definite proof of gonococcal endocarditis is obtained from a positive blood culture, like meningococcal septicaemia the growth of the organism from the blood stream is sometimes difficult. The gonococcal complement fixation test is usually positive.

TREATMENT

Gonococcal endocarditis has nearly always been fatal in the past. The localization of the infection in the urethra, or its systemic spread, probably depends more on the virulence of the bacteria than on the power of resistance of the patient; nonetheless, in the treatment of gonococcal urethritis it is necessary to regard the general health as well as the local lesion. It is doubtful whether the more general use of penicillin in the treatment of the primary infection will diminish the incidence of endocarditis, but when the heart has become involved no form of therapy can claim any great prospect of success

STREPTOCOCCAL ENDOCARDITIS

TERMINOLOGY

The nomenclature applied to this condition is not standard, so that it has been variably described as infective, ulcerative, malignant, subacute bacterial or bacteriaemic, and septic endocarditis, or as endocarditis lenta The term streptococcal endocarditis has the virtue of emphasizing the cause and it is in keeping with the terminology applied to other types of endocarditis and which also implies the aetiology

AETIOLOGY AND PATHOLOGY

The non-haemolytic *Streptococcus viridans* is the causative organism and this may be recovered from the blood stream during life or from vegetations on the heart valve at necropsy. The blood culture may fail to grow the organism at one or more trials and the test needs to be repeated

A diseased heart supplies a suitable nidus for the establishment of streptococcal endocarditis, and, as a rule, some form of congenital heart disease or acquired valvular injury is present. The injured valve is liable to trap and harbour the organism. The *Streptococcus viridans* can usually be isolated from the mouth, especially when dental sepsis is present It is also known that within a few moments of the extraction of septic teeth a streptococcal bacteriaemia of a few minutes'

duration takes place in three-fourths of all cases, and in one-third of those in which extraction is conducted in the absence of obvious gum infection. Nonetheless, although the entry of the organism into the blood stream is essential to the development of streptococcal endocarditis, the presence of a cardiac defect is the primary factor which determines the start of the infection.

Among the varieties of congenital cardiovascular disease which give rise to streptococcal endocarditis, a bicuspid aortic valve assumes great importance because it accounts for about one-fourth of all cases, and because the complication results in about one-fifth of cases presenting this congenital anomaly. Other forms of congenital heart disease liable to this complication are ventricular septal defect (in two-thirds of the cases), Fallot's syndrome (in one-third of the cases), pulmonary stenosis (in one-third of the cases), and patent ductus arteriosus (in one-fifth of the cases) The infection is not uncommon in coarctation of the aorta It rarely occurs in auricular septal defect Mitral stenosis and aortic valvular disease (rheumatic and rarely luetic) are examples of acquired heart disease which not infrequently exhibit streptococcal endocarditis. Very occasionally the heart is unaffected by previous disease.

The vegetations formed in these several conditions are usually large, irregular and friable, and are situated on, or adjacent to, the diseased valve, or on the mural endocardium An interesting feature of a few cases of ventricular septal defect has been the finding of vegetations on that part of the right ventricle upon which the blood stream, projected through the septal opening, impinges.

SYMPTOMS AND SIGNS

The symptoms of streptococcal endocarditis are ushered in gradually and it is some time before the typical clinical picture is established. Lassitude is the first symptom to appear and others are best described under the three headings of septicaemic, cardiac and embolic.

Septicaemic symptoms

Irregular pyrexia is present, as a rule, but rarely the temperature remains normal throughout the illness. Sweating and chills are common, and rigors, although uncommon, may take place Pallor is associated with some degree of anaemia Apart from a diminution in red cells and the haemoglobin content, the blood count shows a slight or moderate polymorphonuclear leucocytosis Petechial haemorrhages should be sought in the conjunctivae, palate, finger-nail beds, retina, and in the skin. The " septic spleen " shows as splenomegaly. Septicaemic effects on the kidneys give rise to pin-head areas of subcapsular haemorrhages (" flea-bitten " kidney) or to haemorrhagic nephritis or to both, the urine will show either frank haematuria or red cells discovered by microscopical examination A blood culture will demonstrate the *Streptococcus viridans* at the first or subsequent attempts, but often the isolation of the organism is difficult. Clubbing of the fingers and toes is a characteristic sign in streptococcal endocarditis Its mechanism may be associated with a prolonged state of toxaemia and is perhaps comparable with the arthropathy which develops in bronchiectasis If the infection occurs in congenital heart disease with cyanosis, clubbing is often already present, but when arthropathy is found in acquired heart disease, streptococcal endocarditis is the usual cause.

Cardiac symptoms

The form of cardiac enlargement and the character of the murmur are peculiar to the valvular injury or congenital anomaly *which prevails before the onset* of the infection, but special circumstances need to be mentioned. Thus, in the rare instances where the infection is unassociated with a cardiac defect, some degree of generalized cardiac enlargement may be present, combined with evidence of heart failure. When the infection has been *superimposed on a bicuspid aortic* valve the systolic and diastolic murmurs of aortic stenosis and incompetence develop in the course of the illness. Murmurs associated with other valvular defects may change slightly in character as the vegetations increase in size and involve other valves Occasionally the infected ductus arteriosus becomes obstructed by the vegetations so that the characteristic machinery murmur is lessened or may disappear.

Embolic symptoms

Emboli, in the course of streptococcal endocarditis, may produce local septic effects in addition to those which result from sudden arterial occlusion at the site. The arteries most liable to embolism with resulting infarction in streptococcal endocarditis are the systemic, cerebral, retinal, renal, splenic, coronary and mesenteric Localization of the infection in the walls of arteries causes weakening and gives rise to aneurysm (mycotic or septic aneurysm) Isolated small painful nodules in the fingers or feet (Osler's nodes) do not last long and usually resolve in a few days.

The association of auricular fibrillation with bacterial (streptococcal) endocarditis is rare, but the explanation of this is only partly known. Bacterial endocarditis is a common complication of congenital heart disease, whereas auricular fibrillation is infrequent. Again, auricular fibrillation is a common event in mitral stenosis and heart failure, but bacterial endocarditis affects the heart in its pre-failure phase and before fibrillation has set in. Thus, congestive heart failure appears to be inimical to the development of bacterial endocarditis. Nonetheless, heart failure may develop during bacterial endocarditis, usually without fibrillation

PROGNOSIS AND TREATMENT

Before the introduction of penicillin, recovery from streptococcal endocarditis was unusual Only isolated cases responded to treatment by a sulphonamide *given in adequate doses and continuously over long periods* Penicillin is still under trial but, so far, results have been encouraging over short periods at least The infection has been ended in nearly one-half of the cases by giving half a million Oxford units of penicillin intramuscularly daily for 28 days Occasionally in aortic valvular disease penicillin treatment, apart from overcoming the infection, has accentuated the aortic leak and this in turn has precipitated left ventricular failure.

If streptococcal endocarditis complicates a case of patent ductus arteriosus, ligation of the ductus is a curative procedure

RHEUMATIC ENDOCARDITIS

Rheumatic valvular disease occasionally appears in its acute form, but usually it either develops during rheumatic fever or subsequently, or the valvular lesion

may present fortuitously or with symptoms of heart failure as a first indication of rheumatic infection in the past. Acute rheumatic endocarditis will be described first, and chronic rheumatic endocarditis will be considered in relation to the valvular lesion it has produced, namely, mitral stenosis, aortic incompetence, aortic stenosis, or tricuspid disease.

ACUTE RHEUMATIC ENDOCARDITIS

(*Synonyms*—Warty, verrucose, simple, granulomatous, or non-bacteriaemic endocarditis)

Rheumatic involvement of the endocardium takes the form of small firm bead-like nodules arranged in chain-fashion along the contact margins of the mitral cusps or along the line of the closure of the aortic cusps, and less frequently, on the tricuspid valve. They may also appear on the chordae tendineae and, more rarely, on the mural endocardium Healing takes place by scarring, which results in thickening of the valves with retraction and shortening of the chordae These changes may be accentuated by recurrent attacks of rheumatic infection.

SYMPTOMS AND SIGNS

The clinical diagnosis of acute rheumatic endocarditis is seldom certain The condition may follow rheumatic fever or may arise apart from it. The patient usually shows restlessness, sleeplessness, shortness of breath, grey cyanosis, a varying degree of pyrexia, and tachycardia Pain may be absent and it is not always dependent on pericardial involvement, although signs of this are commonly present. A mitral systolic murmur, characteristic of mitral disease, is present

It is likely that verrucose endocarditis passes unrecognized in a number of patients, and established mitral valve disease, discovered in the course of a systemic clinical examination, is the first indication that it has been present.

PROGNOSIS AND TREATMENT

When the characteristic symptoms of acute rheumatic endocarditis are all present and are severe, recovery is unlikely, although it sometimes takes place Treatment can only be of the palliative kind and directed to easing the symptoms and giving the patient rest

RHEUMATIC FEVER (ACUTE RHEUMATISM)

When the previous health history of patients with mitral stenosis is traced, there may or may not be a history of rheumatic fever or chorea in childhood.

AETIOLOGY

There is some evidence that a streptococcus is the indirect cause of rheumatic fever and that the pathological changes may represent an allergic response on the part of the tissues to the infection. Absolute proof of this contingency is still missing. Although it has been shown that tonsillitis has no direct bearing on the aetiology of rheumatic fever, the incidence of rheumatic fever appears to keep parallel with the incidence of upper respiratory infection in certain series which have been investigated. There is, however, no justification for tonsillectomy on the grounds that such a procedure might prevent an attack of rheumatic fever. The association between "growing pain" and rheumatic fever is a doubtful one.

The infection is rare in patients under 2 years of age and is uncommon in those below the age of 5 years. From 5 to 15 years of age is the most susceptible period and the first attack of rheumatic fever seldom occurs after the age of 20 years. It affects a higher proportion of girls than of boys. It affects all social classes but it is a little commoner amongst the poorer classes. Although common to all climates it is less rife under warm and dry conditions Similarly, its seasonal incidence varies and is highest during the autumn and winter months.

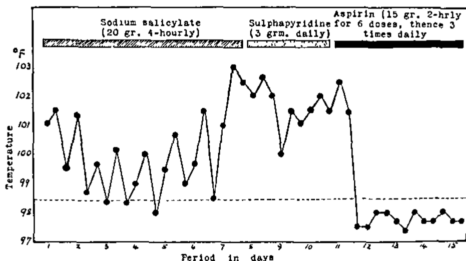


FIG 150—The effect of sodium salicylate, sulphapyridine, and aspirin, on the temperature of a patient with rheumatic fever

SYMPTOMS AND SIGNS

The attack commences with fever and severe joint pain Characteristically, the pain spreads from joint to joint, and usually more than one joint is affected at the one time. The arthritis commonly starts in the knees and in succession might involve ankles, wrists and elbows So severe is the pain that a patient resents any movement. The affected joint is hot and swollen but as a rule it is not red.

Rheumatic nodules may form in connexion with tendons, especially over the extensor aspect of the wrists, elbows and ankles or along the occipital ridge, and sometimes on each side of the thoracic spine. They usually outlast the pyrexial period by many months and they do not, by themselves, possess any special significance, unless it is that the heart is seldom healthy in those patients exhibiting nodules.

When the rheumatic infection appears in the form of *chorea*, coarse, purposeless and involuntary movements of the limbs and face may be confined to one side at the start. Even with treatment the movements may last many weeks and only gradually cease.

TREATMENT

Aspirin is the medicine of choice in rheumatic fever. It is best given as 15 grains two-hourly for six doses and then four-hourly for four weeks, but the nocturnal doses should be omitted after the first week if the patient is asleep. The superiority

of aspirin over sodium salicylate is illustrated in Fig. 150. A sulphonamide is ineffective in reducing the pyrexia of rheumatic fever and it is under trial in prophylactic treatment. Thus, since the incidence of rheumatic fever may have some association with the incidence of upper respiratory infection, and since a sulphonamide might reduce the incidence of such infection, its ability to prevent rheumatic fever is being tested. Even if some success should attend this therapeutic trial, which needs to be adequately controlled, its universal application, if eventually recommended, will prove a difficult problem.

The duration of the rest period prescribed for a child recently convalescent from rheumatic fever has still to be determined. The effect of rest in preventing the onset of endocarditis, or in retarding its progress if it is already present, is not yet known. In the meantime, a reasonable procedure is to allow a patient to be ambulatory within a month of a return of the temperature to normal, provided that there is no contra-indication. There is not any proof that confinement to bed for periods of 12 months or longer is a superior procedure in preventing the onset of endocarditis or to impeding its progress if it is already present

MITRAL STENOSIS

SYMPTOMS AND SIGNS

Symptoms which connote mitral stenosis, the commonest of all valvular affections, usually appear in patients between the ages of 25 and 45 years, but they may also occur at a younger age and even in young children. Routine examination of school-children and of young subjects entering military or other services often discovers the lesion unaccompanied by symptoms. Although rheumatic fever is the only cause of mitral stenosis, neither acute arthritis nor chorea can be elicited in the past health history of about one-half of the cases. Breathlessness is a common presenting symptom when heart failure has set in, and the characteristic signs of the mitral lesion may then be readily made out except in some cases of auricular fibrillation Pain in the left breast may be a troublesome complaint in nervous subjects with mitral stenosis. The pain simulating cardiac ischaemia is often met with, but it differs from the painful syndrome of coronary disease in regards to prognosis, for in mitral stenosis the threat of cardiac infarction is absent The association of cardiac ischaemia in mitral stenosis and a ball or mass thrombus in the left auricle has sometimes been observed (Fig. 151). Tiredness and palpitation are two other symptoms commonly met with especially in nervous subjects with tachycardia

A malar flush with slight cyanosis of cheeks and lips gives to the face of a patient with mitral disease a distinctive appearance (mitral facies), but it can seldom help in early diagnosis for it is not met with until the disease has been present for some time The pulse is small as a rule and the pulse pressure is correspondingly reduced because of a lowered systolic blood pressure. It is often irregular from auricular fibrillation when the clinical diagnosis of the underlying mitral stenosis might be obscured. The apex beat is often displaced outwards because of enlargement of the right heart and left auricle. The extent of the displacement is, therefore, useful in estimating the degree to which these heart chambers are enlarged The beat is both diffuse and forcible if there is much displacement, when the shift is slight, as in early mitral stenosis, it might

still be forcible even in the absence of tachycardia. In mitral stenosis with *auricular fibrillation showing prominent outward displacement of the apex beat,*

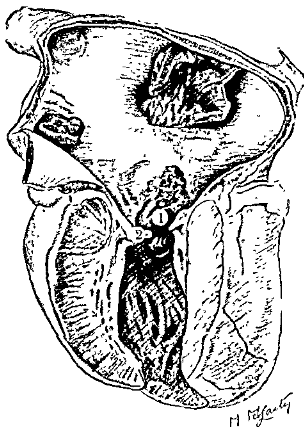




FIG 151.—Mass thrombus (1) in the left auricle in a patient with mitral stenosis (2) who was subject to the pain of cardiac ischaemia during life

pulsation should be sought to the right of the sternum because the extension in this direction of an aneurysm of the left auricle is not rare A thrill in the

mitral area almost always signifies mitral stenosis; its timing in relation to the cardiac cycle has no value because whether presystolic, systolic, or diastolic, it has the same meaning Although the presence of a thrill usually means mitral stenosis, its absence does not exclude the lesion; indeed, it is commonly missing among the signs of mitral disease especially in the early stages.

The clinical diagnosis of mitral stenosis rests chiefly with auscultatory findings A loud first sound in the mitral area is expected unless it is obscured by a systolic murmur; this murmur is moderately loud and is usually a little rough, and sometimes quite rough It is best heard in the reclining posture, with the patient inclined to the left, and after exercise which has induced tachycardia The conduction of the murmur into the axilla is without special significance The features of the innocent mitral systolic murmur have been described on page 31 and will not be referred to here. The remark might be repeated, however, that whenever a systolic murmur in the mitral area has been accepted in any patient as an indication of mitral disease, the diagnosis of mitral stenosis and not mitral incompetence should be applied in that the term mitral stenosis stands for the comprehensive clinical, cardiographic and pathological picture of rheumatic mitral disease, it implies that some degree of mitral incompetence may be present The diastolic murmurs of mitral stenosis are distinctive, in quality they are always rough, they also become louder with tachycardia and when auscultation is carried out with the patient reclining and turned on the left side. The murmur may occur in late ventricular diastole (presystolic or better named *auricular systolic murmur*), when it continues up to the loud ventricular portion of the first heart sound. This murmur disappears at the onset of auricular fibrillation. It is necessary to emphasize that in the differentiation of the presystolic murmur of mitral stenosis from other sounds that simulate it, the common error is to nominate this characteristic murmur when it is absent, and not a failure to find it when it is present Thus, when a doubt exists as to the real nature of the murmur it is unlikely to be the one typifying mitral stenosis The mid-diastolic murmur follows immediately on the third heart sound and it does not follow immediately after the second sound In mitral stenosis the third heart sound is seldom clear on clinical auscultation and in the phonocardiogram it is contiguous with the mid-diastolic murmur which probably always follows it. The second sound in the pulmonary area is often accentuated in long-standing mitral stenosis, but not, as a rule, in early mitral disease, so that it has no value in diagnosis, especially as it is commonly accentuated in health

The *electrocardiogram* (Figs 152 and 153) in a patient with mitral stenosis may show a low voltage curve, slight deviation of the electrical axis to the right, and tall, wide or even bifid auricular waves Prominent axis deviation means added pulmonary incompetence in mitral stenosis or in auricular septal defect. Auricular fibrillation (Fig 154) is common The cardiogram cannot often help in the diagnosis of the early lesion for it is seldom characteristically abnormal.

On *cardioscopy* the changes are characteristic although they vary from patient to patient according to the degree of enlargement presented by the separate moieties of the heart, particularly the pulmonary artery, conus, and the left auricle. Four teleradiograms (Figs 155, 156, 157 and 158) typify these variations in the contour of the heart shadow when viewed in the anterior position. As

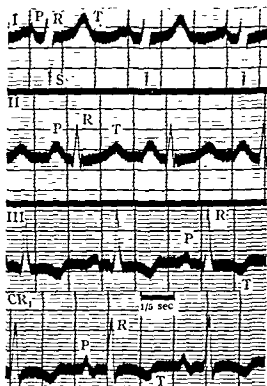


Fig 152 —Mitral stenosis The P wave is broad and bifid in lead I Right axis deviation and right heart preponderance instanced by inversion of the T wave in leads III and CR$_1$, and absence of the S wave in CR$_1$

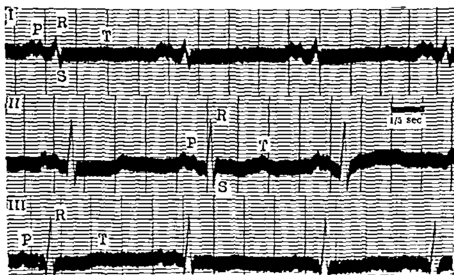


Fig 153 —Mitral stenosis Small voltage in lead I Bifid P waves Low T waves from digitalisation.

a general rule the shadow in the anterior view tends to assume the shape of a square because of the enlargement of the pulmonary arc, left auricular appendage,

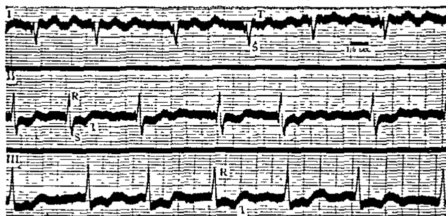


Fig 154 —Auricular fibrillation in mitral stenosis Right axis deviation Depression of S-T segment from digitalisation



Fig 155 —Mitral stenosis Early changes in the anterior view consist of fulness of the pulmonary arc (1) and the conus (2) while the left auricle (3) is seen projecting to the right behind the upper part of the right auricle (4) Male aged 18 years

and conus to form the left upper quadrant, and of the left auricle above the right auricle to form the right upper quadrant Congestion of the pulmonary vessels at the hila appears fairly early. Occasionally the left auricle extends



FIG. 156.—Mitral stenosis. Enlargement of the pulmonary arc (1), conus (2), right auricle (3), and left auricle (4). Early hilar congestion (5). Male aged 38 years.



FIG. 157.—Mitral stenosis. The pulmonary conus or appendage of left auricle (1) is specially prominent. Enlargement of the pulmonary arc (2) and right auricle (3). Much hilar congestion (4). Male aged 38 years.

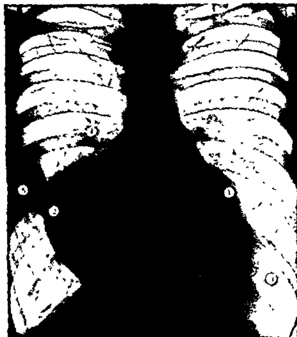


FIG 158.—Mitral stenosis Enlargement of the appendage of left auricle (1) Aneurysmal dilatation of the left auricle (2) Left ventricle (3) displaced to left Hilar congestion (4) Interlobar effusion (5)



FIG 159.—Mitral stenosis The left auricle impression (1) in the left oblique position is seen as a comma-like curve, and is placed immediately below the left bronchus impression (2).

far to the right and this aneurysmal dilatation causes it to approach the right side of the chest. The outer border of the left ventricle may reach far to the left in mitral stenosis, but this is on account of displacement by the enlarged right heart and not the result of left ventricular hypertrophy. The left oblique position will show enlargement of the body of the right ventricle and the left auricle impression is seen as a comma (Figs. 159 and 160). In the right oblique view the left auricular impression is specially prominent (Figs. 161 and 162); at the



FIG 160 —Mitral stenosis Distended left auricle produces a comma curve (1) in the left oblique view Left ventricle (2) Right ventricle (3) Aortic arch impression (4) Left pulmonary artery (5) Supra-aortic triangle (6) Patient was a male aged 55 years

level of this impression the heart shadow is deep owing to a prominence of the conus and pulmonary arc on the opposed side Sometimes (Figs. 163 and 164) the left auricle impression in the right oblique view is absent because the oesophagus has slipped to the left from the summit of the distended left auricle, and in this event the barium swallow is deviated to the left instead of to the right in the anterior view (Fig 165) This slipping of the oesophagus is naturally more common in cases showing great distension of the left auricle, but it sometimes takes place in early enlargement of the auricle, in which case its recognition is of greater importance on that account.



FIG 161—Mitral stenosis Prominent left auricle impression (1) in the right oblique view Aortic arch and left bronchus impressions (2) are fused Female aged 43 years



FIG 162—Mitral stenosis Prominent left auricle impression (1) in the right oblique view Aortic arch and left bronchus impressions (2) are fused Male aged 44 years



FIG. 163—Mitral stenosis in right oblique position showing absence of left auricular impression from slipping of the oesophagus Barium swallow takes a straight course after forming the aortic arch (1) and left bronchus impressions (2) Summit of left auricle indicated by arrows



FIG 164—Mitral stenosis in right oblique position showing slipping of oesophagus from summit of left auricle (1) making the left auricular impression (2) less deep

DIAGNOSIS OF EARLY MITRAL STENOSIS

The description of mitral stenosis already given concerns the condition when it is fully developed, but it is the diagnosis of the early lesion that presents the greatest difficulty, and the following remarks are intended to diminish this

A past history of any of the manifestations of acute rheumatism does not materially help in deciding an early affection of the mitral valve Indeed, the past history may hinder a proper assay of the clinical signs. No such history

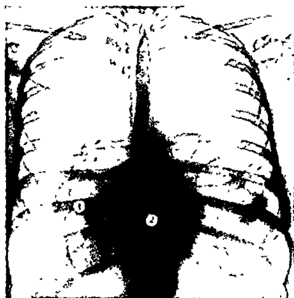


FIG 165 —Aneurysmal dilatation of the left auricle (1) in mitral stenosis showing a reversed left auricular impression (2) in the anterior view from slipping of the oesophagus

can be obtained in about one-half of the cases of well-established mitral stenosis. The mitral facies is not expected until the condition has been present for a number of years. The pulse tends to be small early on and this serves as a useful clinical sign, although it has many exceptions. The systolic blood pressure as a rule is somewhat lower than the average value in subjects of the same age, producing a smaller pulse pressure, but this again has notable exceptions The apex beat is often forcible and this finding has greater significance when the heart rate is not rapid Similarly, a loud first sound in the mitral area is more significant when tachycardia is absent The diagnosis of mitral stenosis cannot rest on the finding of a loud first sound because this is so common in health Triple rhythm due to the addition of the third heart sound is common in mitral stenosis and it is usually conjoined with a mid-diastolic murmur which immediately follows it. A systolic murmur in the mitral area is usually the first reliable clinical sign to appear in mitral stenosis The murmur is moderately loud and rough or roughish in character ; it is best heard in the reclining posture, although it is audible in the upright posture as well , it may be less intense on deep inspiration, but it never disappears during this manoeuvre If the murmur is rough, auscultation after

induced tachycardia with the patient inclined to the left side may bring out a presystolic murmur. This rough systolic murmur in the absence of hypertension, aortic incompetence, aortic stenosis, or heart enlargement from heart block, should indicate mitral stenosis. Of greatest importance in the auscultatory diagnosis of mitral stenosis in a patient with a mitral systolic murmur is the finding of a mid-diastolic murmur, for this is an invariable finding in the phonocardiogram. One should, therefore, strain to hear it in any suspected case, although clinical auscultation may fail at the end to elicit it. The intensity of the second sound in the pulmonary area is no aid to the recognition of early mitral



FIG 166.—Oesophageal impressions in left oblique view in a young healthy subject. The aortic arch impression (1) has its concavity to the right, this diminishes the left bronchus impression (2) Thence the stream is straight or "plumb-line", showing no left auricular impression

disease. As a rule, the *electrocardiogram* is physiological although a prominent P wave or slight prolongation of the P–R period is sometimes observed. *Cardioscopy* is invaluable in the diagnosis of early mitral stenosis, although it is not without its difficulties. In the anterior view, although the pulmonary arc may appear full, localized prominence of the conus of the right ventricle gives the best clue. The diagnosis is next tested by watching the progress of the barium swallow in the oblique positions. In the right oblique position, after tracing the aortic arch and left bronchus impressions, the barium stream should be carefully watched as it commences to form the left auricular impression, for in the pathological curve the stream changes its course abruptly to the patient's right as it passes downwards. The impression is not expected to be very deep in the early

case, but a support for the diagnosis may come from finding a deeper cardiac shadow at this level. In the left oblique position a comma curve is a normal finding in healthy children and in young subjects, but it is often absent in healthy adults. It is never absent in mitral stenosis. Whenever the stream is directed straight down (plumb-line) (Fig 166), the absence of mitral stenosis is certain, and therein lies the value of this test.

Course of the disease and its complications

The course of mitral stenosis depends on its degree, the involvement of other valves, on the stresses which the patient faces, and these relate to age, sex and occupation, and on the incidence of complications. Thus, the outlook deteriorates if aortic incompetence or tricuspid damage is added, although in the latter condition, for a time at least, the late symptoms of mitral stenosis, notably ascites and oedema, may prove to be less prominent. In children and young adults the prognosis is poor if mitral stenosis is severe, and the outlook is similarly ominous in those following occupations which involve manual labour, and in female patients who undergo the stresses incurred by successive pregnancies involving the care of a family. A young patient with mitral stenosis is subject to illnesses which are so much a part of the condition as not to be regarded as complications because a patient cannot escape many of them in the course of the disease. These clinical states will be enumerated here, although most of them are described more fully elsewhere.

Recurrent attacks of rheumatic fever.—A patient who has suffered from rheumatic fever is susceptible to further attacks, but this susceptibility diminishes with age, so that recurrence after the age of 25 years is uncommon. When rheumatic fever is seen in patients of between 20 and 25 years of age it will be a recurrent condition in the majority of cases because it is unusual for the first attack to happen after the age of 20 years.

Recurrent attacks of acute bronchitis.—The frequency with which paroxysms of acute bronchitis supervene in mitral stenosis deserves more attention. The event is too often attributed to an exacerbation of heart failure. Such patients, especially in winter, develop increased breathlessness (often severe), cough, sputum (usually thin and often coloured with blood), and on auscultation show a prolonged expiratory phase, crepitations and rhonchi. Cardioscopy provides evidence of emphysema and bronchitis as well as changes in the heart typifying mitral stenosis. The recognition of this syndrome serves to direct treatment to the bronchitis while maintaining digitalisation at the same dosage as before the onset of the breathless paroxysm.

In connexion with these pulmonary symptoms it should be remembered that the association of pulmonary tuberculosis and mitral stenosis, although known, is uncommon.

Intracardiac thrombosis.—Thrombosis within the heart is commonplace in mitral stenosis. If this has taken place in the right heart, pulmonary embolism and infarction may result. If situated in the left side, systemic embolism, especially cerebral, is a common event. Allusion has already been made to the probable association in some patients of a ball or mass thrombus in the left auricle and pain of cardiac ischaemia.

Streptococcal endocarditis.—Ulcerative vegetations on the mitral valve in mitral stenosis and bacteriaemia from infection by the *Streptococcus viridans* usually take place when the mitral injury is an early one. Streptococcal endocarditis is rare in mitral stenosis with heart failure and auricular fibrillation.

Heart failure.—Breathlessness, and pulmonary congestion when examined by cardioscopy, appear in mitral stenosis a long time before the onset of auricular fibrillation and the more obvious signs of right heart failure.

TREATMENT

In a patient recovering from acute rheumatism or chorea there is no good evidence to show that rest in bed for periods of six months or longer can prevent involvement of the mitral valve or limit the spread of the process when it has already commenced. There is not any evidence either that tonsillectomy in a child subject to recurrent tonsillitis reduces the incidence of mitral stenosis, although the operation may be amply justified on other grounds.

When mitral stenosis has become established, the patient requires supervision rather than treatment during the period prior to the onset of complications, and re-examination at regular intervals is to be encouraged, although care must be taken to lift any undue worry the patient may have about his or her condition. In respect of physical activities, exercise may be permitted and exertion should be prohibited, so that a younger subject needs to be advised about a prospective career and directed into one of a sedentary or clerical kind. The adult patient should be advised to move out of any heavy employment he or she may be following. When pregnancy has taken place in a patient with mitral stenosis special supervision is necessary and this is described elsewhere (*see* page 256). Complications associated with the lesion require that form of treatment which is customarily assigned to them.

TRICUSPID STENOSIS

Although tricuspid stenosis is not a rare finding at necropsy in cases of rheumatic endocarditis, it is only infrequently diagnosed during life. This is due to a neglect to seek in a patient with mitral stenosis the added signs of tricuspid involvement. Tricuspid stenosis is present in about one-fifth of the cases of rheumatic heart disease, but it is in less than one-tenth of these cases that clinical diagnosis is possible.

SYMPTOMS AND DIAGNOSIS

Many of the symptoms of tricuspid stenosis are common to mitral stenosis, which is always present, but a few are characteristic of the tricuspid lesion. Dyspnoea is often less conspicuous, so that a patient with mitral stenosis, even with ascites, may be able to sleep in the reclining posture if tricuspid stenosis is also present. A combination of cyanosis and jaundice sometimes gives the face a distinctive colour. Distended veins in the neck, an expression of a raised venous blood pressure, may show systolic pulsation. Similar pulsation may also be observed in an enlarged liver, although care is necessary to distinguish this from pulsation transmitted from an enlarged right heart which is a common sign. Ascites and oedema of the lower extremities are also common to tricuspid

stenosis. The pulse is characteristic of mitral stenosis, so that as a rule it is small and is often irregular from auricular fibrillation. In the heart three characteristic signs should be sought in addition to those which are the outcome of mitral stenosis. Thus, the right auricle is greatly distended so that its pulsation may be felt to the right of the sternum. A thrill is sometimes present in the tricuspid area, especially in young subjects, and a systolic murmur is heard in the same area. A mitral systolic murmur from mitral stenosis is heard faintly some distance to the right of the mitral area, but in the presence of tricuspid stenosis the murmur gains in intensity as the tricuspid area is reached.

The *electrocardiogram* is not distinctive but it is more certain to show changes characteristic of mitral stenosis.

In addition to the typical findings of mitral stenosis, *cardioscopy* supports the diagnosis of associated tricuspid stenosis if there is conspicuous enlargement of the right auricle and if the pulmonary congestion is not excessive.

PROGNOSIS AND TREATMENT

Since it is true that the more severe the infection in acute rheumatism affecting the heart, the higher is the incidence of tricuspid stenosis, the outlook is grave in young patients. In older subjects, however, the mechanical effects of the tricuspid deformity are a slight advantage in mitral stenosis, not that they prolong life, but in that invalidism due to mitral stenosis is to some extent alleviated; it is thought that this improvement is the result of lessened pulmonary congestion and right ventricular enlargement, which are the inevitable effects of mitral stenosis.

Treatment should follow that indicated in mitral stenosis, so that in the presence of auricular fibrillation continuous digitalisation must be maintained. Since ascites and oedema are obvious signs when tricuspid stenosis becomes added in a patient with mitral stenosis, a more regular use of mercurial diuretics is usually necessary.

AORTIC STENOSIS

Thickening of the aortic cusps from any cause will give rise to some degree of obstruction to the outflow of blood through the aortic opening; the extent of this obstruction may vary from slight to extreme, and produce corresponding effects on the heart.

AETIOLOGY

Rheumatic fever is a common cause of aortic stenosis in young subjects when mitral stenosis may also be present. This variety is commonly accompanied by clinical evidence of aortic incompetence.

In elderly subjects *atheroma* is a common cause, and calcification frequently supervenes converting the cusps into a rigid ring. The early part of the aorta may be relatively free from atheroma and a little dilated, so that it is likely that both atheroma and calcification in this instance have been laid down in a valve previously injured by some unspecified infection. This type may be present in young subjects as well and it may be congenital.

Syphilitic endocarditis is not a common cause of aortic stenosis, but in a certain proportion of cases of syphilitic aortic incompetence a stenotic effect is introduced although valvular incompetence is the preponderating lesion.

Congenital aortic stenosis in which the thickening is confined to the cusps is probably not a rare condition ; congenital subaortic stenosis with narrowing at the neck of the left ventricle is rare.

Streptococcal endocarditis can by itself cause aortic stenosis when it supervenes in a patient with a bicuspid aortic valve, or it may accentuate the obstruction already present from some other cause.

SYMPTOMS AND DIAGNOSIS

A patient in whom the aortic narrowing is not severe is usually without symptoms and the lesion is then discovered in the course of routine clinical examination. Again, it may be found as part of the larger picture of rheumatic heart disease. If the stenosis is severe or associated with aortic incompetence the symptoms of heart failure may appear. Giddiness and faintness are occasional complaints, but sometimes the more serious symptoms of streptococcal endocarditis may be the first to direct attention to the heart.

If aortic stenosis is present in childhood, physical development may be retarded, but this and other signs to be described depend on the severity of the aortic narrowing. The pulse may be small and of the anacrotic type. The systolic blood pressure is low, producing a small pulse pressure, but if the aortic incompetence is a conspicuous associated lesion, as it might be, the pulse is large. If aortic incompetence accompanies aortic stenosis, prominent arterial pulsation is noticeable in the neck and sometimes a characteristic *carotid shudder* is present. A systolic thrill is often present in the aortic area. A thrill above the right clavicle is a sign of aortic stenosis only when it is long and associated with a systolic murmur in the aortic area, for a short thrill in the neck is common in young healthy subjects. The apex beat is displaced outwards, especially in the presence of aortic incompetence. A rough systolic murmur in the aortic area is conducted along the vessels of the neck The same systolic murmur, heard in the mitral area, is often as loud and sometimes even louder. A distinct second sound is often heard in the aortic area in aortic stenosis, but in some cases it either is inaudible or is replaced by the diastolic murmur of aortic incompetence, which both here and in the mitral area is characteristically rougher than that in lone aortic incompetence Although it is often impossible to elicit even on direct auscultation in aortic stenosis the early diastolic murmur of aortic incompetence, *such a murmur is often recorded in the phonocardiogram*

Although the four cardinal signs, anacrotic pulse, displaced apex beat, systolic aortic thrill and rough murmur, are sometimes demanded for a diagnosis of aortic stenosis, insistence on these signs being present would render the clinical diagnosis uncommon Indeed, if a rough systolic murmur is well heard in the aortic area the presence of some degree of aortic stenosis has been substantiated. Occasionally when aortic stenosis is severe and left ventricular failure is present, both murmur and thrill may disappear from the aortic area, although the murmur, and even a thrill, may persist in the mitral area ; in this event the finding of a short early diastolic murmur will help to provide the diagnosis.

The *electrocardiogram* is sometimes unaltered or only shows deviation of the electrical axis to the left; usually the tracing is distinctive (Fig 167), showing inversion of the T wave in leads I and II or in II and III in which the descent of the wave is gradual from R–T depression and the ascent steeper. The T wave is

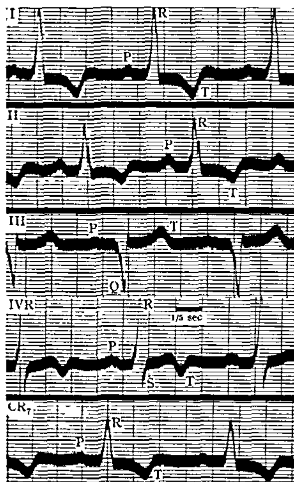


FIG 167—Aortic stenosis Left axis deviation Left ventricular preponderance as instanced by inversion of the T wave with R–T depression in leads I and CR_7, the inversion is deeper in CR_7 than in IVR

also inverted in the chest lead CR_7 These changes depend on the severity of the stenosis and the degree of left ventricular enlargement

At *cardioscopy* varying degrees of left ventricular enlargement will be found depending on the extent of stenosis and especially on the accompanying aortic incompetence. Characteristically in the anterior view, the aorta is curved like a banana because of an outward bowing of the ascending part of the aorta and a

prominent projection of the aortic knuckle upwards and to the left (Fig 168). In the presence of aortic incompetence this part of the aorta will show conspicuous pulsation. In the rheumatic variety the signs of mitral stenosis are often present There may be calcification of the valve.

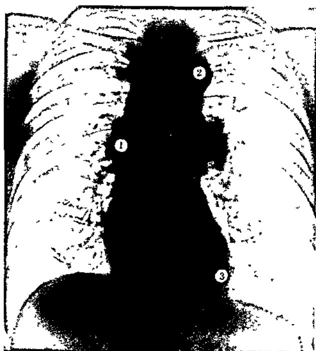


FIG 168 —Aortic stenosis Ascending aorta (1) prominent to the right, and upward and outward displacement of the aortic knuckle (2) The left ventricle (3) is hardly enlarged

PROGNOSIS

In general it may be said that aortic stenosis has a better outlook than any of the valvular lesions and is best when atheroma is the cause, or when it is of congenital origin and the stenosis is slight Prognosis is less favourable when the stenosis is severe or when aortic incompetence accompanies it, and especially if this has resulted from syphilis.

AORTIC VALVE SCLEROSIS

In elderly subjects it is common for atheroma with calcification to form deep in the sinuses of Valsalva and to extend on to the aortic surface of the aortic cusps. Such a change causes neither aortic stenosis nor incompetence, and is immaterial in prognosis. Its importance lies in its physical signs. It produces a systolic murmur which is best heard in the mitral area, but which is conducted towards the aortic area ; it is characteristic of this murmur that it can be heard distinctly towards the aortic area in the upright posture on direct auscultation, even though inaudible towards that area in the reclining posture during auscultation with a stethoscope.

AORTIC INCOMPETENCE

Unlike mitral incompetence a diagnosis of aortic incompetence is significant by itself, for enlargement of the left ventricle prefacing the onset of left ventricular failure is common to all conditions which cause it ; its progress, however, is closely associated with the nature of the valvular injury, so that it is always desirable to include the aetiology of the lesion in the clinical diagnosis

AETIOLOGY

Rheumatic fever is the most common cause of aortic incompetence. Aortic stenosis often accompanies rheumatic aortic incompetence. It is also frequently accompanied by mitral stenosis and sometimes by tricuspid stenosis as well. In young subjects, when aortic incompetence is prominent, clinical evidence at least of mitral stenosis is often wanting, and even on cardioscopy it is difficult to be sure of the presence of left auricular distension from mitral stenosis. The clinical diagnosis of *lone* aortic incompetence is, therefore, warranted in these cases.

Syphilis is the next commonest cause of aortic incompetence. The leak results either from thickening and retraction of the aortic cusps due to inflammation, or from dilatation of the aortic ring caused by aortitis, the former kind is three times as common as the latter. In the *first type* the aortitis involving the early part of the aorta spreads to the cusps to produce valvulitis and aortic incompetence early on. Aortic incompetence is inimical to the formation of an aneurysm so that such a change is uncommon in this variety. The coronary orifices are involved early, giving rise to cardiac ischaemia. Some degree of aortic stenosis is occasionally met with in this type In the *second type*, aortitis placed more distally in the aorta gives rise to aneurysmal dilatation which extends to the aortic ring, producing aortic incompetence later, but without causing much injury to the cusps. Cardiac ischaemia is also a late symptom from eventual involvement of the coronary orifices Aortic stenosis is not met with in this type.

When *atheroma* of the aortic valve produces aortic stenosis some degree of incompetence is common, but it is rarely the chief lesion, and it is often difficult to discover clinically, although its presence may be proved phonocardiographically.

Streptococcal endocarditis, superimposed on a diseased aortic valve, may increase the degree of incompetence. When previous clinical examination has found no aortic murmurs, the finding of aortic incompetence (usually accompanied by stenosis) in the course of bacterial endocarditis is presumptive evidence that *the infection has started in a congenital bicuspid aortic valve.*

Aortic incompetence sometimes develops in the course of *hypertension* (relative incompetence), when cardioscopy will show bowing of the first part of the aorta from elongation which also causes the aortic knuckle to project upwards to the level of the inner end of the left clavicle. Although there is slight dilatation of the aorta, necropsy may show that atheroma of the cusps has often contributed to the incompetence. Aortic incompetence is not uncommon in the hypertension of coarctation of the aorta

When aortic incompetence either develops, or increases suddenly, following the *rupture of a cusp*, bacterial endocarditis is usually present. It is unusual for rupture to take place in any other disease of the valve or in a healthy valve.

Fenestration, or some other congenital defect of the aortic cusps, is a very rare cause of aortic incompetence. An aneurysm of the sinus of Valsalva may rupture and form a fistulous opening, joining the aorta to the pulmonary artery or to the right heart.

SYMPTOMS AND SIGNS

A sensation of throbbing in the neck and head and momentary faintness are the less severe symptoms of which a patient with aortic incompetence may sometimes complain. The characteristic pain of cardiac ischaemia is occasionally present; although this symptom can occur in rheumatic aortic incompetence, especially when accompanied by mitral stenosis, syphilitic aortic incompetence is the most likely diagnosis. Indeed, it is a general rule that when a patient with aortic incompetence has become subject to the painful paroxysms of cardiac ischaemia, syphilis is the cause. Increasing breathlessness heralds the onset of heart failure, which usually takes place with normal heart rhythm, and in the form of paroxysmal nocturnal dyspnoea (cardiac asthma)

The pulse is collapsing in character (unless in the presence of aortic or mitral stenosis), and the pulse pressure is large from a lowering of the diastolic pressure, and to a lesser extent, from a raising of the systolic pressure. Arterial pulsation is usually prominent wherever peripheral arteries can be inspected or felt. This pulsation may be seen in the radial, brachial, and carotid arteries, and in the retinal arteries on retinoscopy, it is prominent in the femoral arteries where the blood pressure is raised to a higher value than in the arm. This increased pulsation can also be demonstrated in accessible capillary beds.

The apex beat is displaced outwards and is usually diffuse and forcible. Rarely, a diastolic thrill is present in the aortic area, when accompanying aortic stenosis is the cause. On auscultation in the mitral area a systolic murmur is never missing, although it is not loud when the lesion is early, and a diastolic murmur follows immediately after the second sound; this murmur is soft if there is only incompetence, but when aortic stenosis is also present the murmur assumes a slightly rough character. Attention to these signs makes it possible to diagnose aortic incompetence by auscultation in the mitral area alone In the aortic area the same diastolic murmur is audible, but as a rule it is better heard lower down and frequently to the left of the midline In this connexion it should be remembered that the commonest organic murmur to be elicited in the pulmonary area is the diastolic murmur of aortic incompetence. The side on which the murmur is louder can in no way determine the cause of the incompetence. Should there be doubt about the presence of the diastolic murmur, and this may happen when the lesion is early or when the second sound is well heard, direct auscultation must be carried out with the ear over the lower part of the sternum and with the patient in the upright position holding his breath at the end of expiration. When rheumatic fever has caused aortic incompetence it may be difficult to decide whether the systolic murmur in the mitral area is part of aortic incompetence or whether it indicates mitral stenosis. In syphilitic incompetence when the leak is fairly severe, splitting of a loud and rough first heart sound often associated with a systolic murmur, may simulate the presystolic murmur of mitral stenosis (Austin Flint murmur), in fact this auscultatory sign is no different to that found in other

clinical states which produce enlargement of the left ventricle, notably hypertension. Again, the presystolic murmur might indicate associated mitral stenosis, or the early diastolic murmur might continue up to the first heart sound creating the impression of a presystolic murmur.

Although the *electrocardiogram* may be normal, characteristically it shows a deviation of the electrical axis to the left and inversion of the T wave in leads I and CR_7, and often in lead II as well, with R–T depression.

Cardioscopy shows enlargement of the left ventricle and prominent pulsation in the aorta; the systolic stroke of the heart is usually large. Other changes may be present and permit a diagnosis of the cause (Figs 169 and 170).



FIG 169 —Aortic incompetence Great enlargement of the left ventricle (1) and hilar congestion from failure (2) The aorta which showed increased pulsation on cardioscopy is not much altered in contour, but this does not exclude aortitis as the cause

COURSE AND PROGNOSIS

The ultimate outlook in aortic incompetence is ominous, but the prognosis needs to be assessed in individual patients after certain circumstances, among them age, occupation, aetiology, presence of complications, and extent of the lesion, have been considered. Aortic incompetence in a child is of serious import An occupation involving heavy manual work or one which does not permit adequate rest, will influence unfavourably the course of the condition. In general the syphilitic lesion is more serious than the rheumatic, and aortic incompetence from atheroma carries the best prognosis. The onset of heart failure or streptococcal endocarditis is a grave event It is often difficult to assess from clinical examination

the *extent of the reflux in aortic incompetence* Neither the intensity of the murmur nor the value of the pulse pressure can indicate how great the leak is; the size of the left ventricle serves as the best guide.

DIAGNOSIS

Aortic incompetence as a well-established and a lone condition is not difficult to recognize. Difficulty arises in the diagnosis of the early lesion, and when other conditions such as mitral stenosis and auricular fibrillation obscure it. In regard to early aortic incompetence it is necessary to listen attentively for the characteristic murmur even when the second sound in the aortic area is audible or loud, and here, as well as in the presence of associated lesions, it is important to seek the murmur by direct auscultation under the conditions already enumerated.



Fig 170 —Aortic incompetence Enlargement of the left ventricle (1) Aneurysmal dilatation of the ascending (2) and descending aorta (3) confirms the luetic origin of the incompetent valve

Concerning the aetiology of aortic incompetence there is generally not any difficulty. In a young subject rheumatic fever is the usual cause In an adult the finding of mitral stenosis alongside the aortic leak again identifies rheumatic fever as the cause; in the absence of mitral disease it may be due to syphilis when evidence of aortitis may present at cardioscopy and the Wassermann reaction may prove to be positive. Should aortic stenosis in the adult or elderly patient appear to be severer than incompetence, atheroma is the likely cause. At any age the aortic cusps might be thickened and even calcareous without mitral stenosis or aortitis, when the aetiology is obscure.

TREATMENT

When the syphilitic nature of aortic incompetence has been established, anti-syphilitic treatment needs to be initiated Even though the valve injury is already serious, specific therapy may be worth while in order to prevent the onset of the pain of cardiac ischaemia or to relieve it if it has already set in For this purpose a mixture containing 5 grains of potassium iodide and 1 drachm of liquor hydrarg perchlor. should be prescribed twice daily for twelve months No special advantage can be claimed for arsenical and bismuth preparations over mercury and iodide in the treatment of aortitis

In other forms of aortic incompetence a limitation of the patient's activities is all that is necessary When the symptoms are light, active therapeutic measures need not be applied until the onset of breathlessness, marking the advent of left ventricular failure, indicates rest and the regular use of mercurial diuretics and digitalis.

CHAPTER 8

AORTITIS

THE SITE of election of syphilitic aortitis is the ascending aorta, and especially in its commencement In decreasing frequency it involves the arch, the descending thoracic aorta, and the abdominal aorta. Usually the aortitis is not limited to one of these moieties, but involves the adjacent segments as well. Patchy involvement of the whole thoracic aorta is frequent. Since the site and extent of the lesion decide the symptomatology and course of the disease, it follows that the condition manifests itself clinically in diverse ways.

SYMPTOMS AND SIGNS

According to their symptomatology patients with aortitis can usually be classed in five groups, but when the condition is of long standing, symptoms common to two or even three of the groups may be present in the same patient.

Aortitis without pain

When the lesion is early, localized, and not seriously affecting the root of the aorta, the patient may remain free from symptoms. It is only when such a patient is examined radiologically that aortitis is discovered, although it might evade detection even then When hypertension has been excluded as a cause of the aortic changes, the influence of medial degeneration in producing deformity of the aortic outline must also receive consideration before the diagnosis of aortitis is made In the radiological diagnosis of syphilitic aortitis attention should be directed to the following criteria

1. When hypertension is absent and medial degeneration is not considered to be the cause of the deformity, a localized pulsatile bulge in the ascending aorta, visible in the anterior and left oblique positions, is good evidence of aortitis Prominent localized aortic pulsation has greater significance in the absence of aortic incompetence.

2. If aortic incompetence is present in an adult, and the aorta on radiological examination is suspected to be affected by syphilis, and mitral stenosis is absent, the diagnosis of aortitis becomes more certain.

3. When the patient is rotated to the right through an angle of 45° (left oblique position), if the outer border of the ascending aorta reaches or projects beyond the heart border (represented in this position by the right ventricle), and hypertension is absent, aortitis is the usual cause of the deformity (Fig 171)

4 Although the ascending portion of the aortic arch affected by syphilis presents a "clubbed" appearance (Fig 172) in the right oblique position, a similar change can sometimes result from medial degeneration.

5 The descending aorta may appear prominently on the left in the anterior view from elongation in hypertension or atherosclerosis, but if the aortic outline



FIG 171 —Early aortitis Prominence of the ascending aorta (1), which in the left oblique position reaches the perpendicular drawn at the outer border of the right ventricle (2).



FIG 172 —Aortitis of the ascending limb of the aortic arch produces a clubbing effect (1) in the right oblique position

is irregular, or if it projects to the left early in its course and near the aortic knuckle, aortitis is the likely cause.

6. When the barium-filled oesophagus is examined radiologically in the three orthodox positions, the diameter of the aortic arch is represented by the length of the impression made on the oesophagus by the aortic arch, provided that the impression is seen as a complete semicircle (Fig. 173). It may also be represented by the distance from the deepest point of the aortic arch impression to the opposed border of the aortic shadow (Kreuzfuchs' measurement), although the true opposed border is often difficult to define (Fig 174) These measurements may be greatly increased by dilatation of the aortic arch from syphilitic aortitis.

7. Displacement of the oesophagus in its lower moiety may prove to be the first sign of aortitis affecting the descending thoracic aorta. Although this displacement may be evident in all three positions, it has its optimal viewpoint in one position, usually the right oblique.

Aortitis is commonly found in patients examined by cardioscopy 15 years after the initial syphilitic infection, but the finding is not rare when less than 10 years have elapsed Thus, if an early diagnosis of aortitis is sought, the aorta should be first examined radiologically 5–10 years after the primary infection, but it needs emphasis that aortitis might be present without distorting the outline of the aorta or producing a deformity which will show on radiological examination

Along with these radiological criteria other clinical evidence of syphilis or a positive Wassermann reaction will prove of value in establishing more certainly the presence of syphilis of the aorta, although a negative serological test should not weigh heavily in diagnosis

Aortitis with pain

A constant dull aching pain in one side of the chest, not necessarily induced by exercise although accentuated by it, and often influenced by posture, especially in bed at night, and occurring in an adult, should point to aortitis as the likely explanation if disease in the lung and pleura has been excluded. Here again, radioscopy of the aorta provides the only certain method of deciding the true nature of the underlying condition and even this may fail. If iodide and mercury therapy relieves the pain it supplies added evidence of the syphilitic nature of the lesion.

Aortitis with aortic incompetence

Many patients with syphilitic aortitis are examples of aortic incompetence from the beginning of their illness Tiredness, weakness, and palpitation may be early symptoms, and breathlessness from heart failure may appear later. The presenting signs are those common to aortic incompetence from any cause *Cardioscopy may show aortitis, but in the absence of radiological changes* due to syphilis, failure to demonstrate enlargement of the left auricle from mitral stenosis suggests that rheumatic fever has not been the cause of the aortic incompetence, but not in a young subject. Since syphilis of the aorta commonly affects its root, the inflammation often extends into and retracts the cusps of the aortic valve, or involves the aortic ring, giving rise to dilatation. In either



FIG 173 —The aortic arch impression (1) from a patient with hypertension in the right oblique position, commences at the summit of the aortic shadow and appears to end at its inferior border; the height of the impression in this instance is probably a measure of the diameter of the aorta Right pulmonary artery (2) Left bronchus impression (3) Left auricle impression (4)



FIG. 174 —Close-up view of aortic arch impression (1) in a female aged 57 years, with hypertension Deep x-ray penetration shows the left border of the aortic arch (2) within the shadow of the descending aorta (3) Such penetration prevented a false Kreuzfuchs' measurement being recorded

event aortic incompetence results Once aortic incompetence has occurred aneurysm does not form. Further, when aortic incompetence is primarily due to involvement of the valve, aneurysm is seldom present, but when the incompetence has resulted from a widening of the aortic ring, aneurysm is usually present. It follows that involvement of the cusps usually occurs at an earlier date in the progress of the aortitis than does dilatation of the aortic ring Two distinct anatomical and clinical types of aortic incompetence may thus be recognized

Aortitis with cardiac ischaemia

The presence of syphilitic aortitis may first become known by the appearance of the pain of cardiac ischaemia. Apart from arteritis within areas of syphilitic myocarditis, syphilis of the coronary arteries is confined to implication of the orifices by aortitis. Cardiac ischaemia is not a feature of syphilitic aortitis unless there is stenosis of one or both coronary orifices. Complete closure of one coronary orifice may, however, be present without pain. Cardiac ischaemia due to syphilitic aortitis differs from that caused by coronary atheroma in certain important respects Aortic incompetence is common in syphilitic cardiac ischaemia, so that if aortic incompetence is discovered in a patient the subject of cardiac ischaemia, aortitis is the likely cause. There is not, however, any evidence to show that the added effect of aortic incompetence decides the onset of cardiac ischaemia in a patient in whom the coronary orifices are stenosed from aortitis. In a number of patients antisyphilitic remedies have brought about a temporary or permanent relief from the painful attacks. Coronary thrombosis seldom, if ever, results from stenosis of the coronary orifice by aortitis, and cardiac infarction takes place in the absence of coronary thrombosis. Thus, when infarction has resulted, recovery is unlikely.

Aortitis with aneurysm

The symptoms of an aortic aneurysm compel patients in this group to seek medical examination. Before discussing aortic aneurysm it is expedient to define certain terms in common usage to describe the different kinds of vascular swellings

A *true aneurysm* is a dilatation of the lumen of an artery, and is bounded by the arterial wall. A *false aneurysm* is an apparent swelling of an artery caused by blood that has escaped through a rupture and has formed a mass bounded by tissues outside the arterial wall A true aneurysm may later become a *mixed aneurysm* (true and false) from rupture of its dilated wall. A *cirsoid aneurysm* is a true aneurysm in which the artery is tortuous as well as dilated A *dissecting aneurysm* is an expansion of the arterial wall formed by blood which has passed from a rupture into the wall and has extended within the media or between the media and the intima or adventitia. A dissecting aneurysm may complicate a true aneurysm. An *arterio-venous aneurysm* is the result of a communication between an artery and a vein The varieties of arterio-venous aneurysm known as *aneurysmal varix* and *varicose aneurysm* are direct ruptures of adjacent arteries and veins, caused almost invariably by trauma, but sometimes by inflammation It is clear then that all varieties of acquired aneurysm other than primary true aneurysm (true aneurysm, cirsoid aneurysm and mixed aneurysm) are merely expressions of arterial rupture.

The precise stage at which a localized bulge of the aortic wall should assume the designation of aneurysm cannot be defined. Certainly it merits this description when its tumour-like character is demonstrated by symptoms caused by its pressure on adjoining structures Nonetheless an aortic aneurysm of moderate dimensions may be so situated as not to produce any symptoms from pressure and its presence may only be discovered radiologically. The recognition of aortic aneurysm depends on its situation and on the nature of the injury which it causes to adjacent structures in the course of its extension.

SYMPTOMS AND DIAGNOSIS OF AORTIC ANEURYSM

Aneurysm of the *ascending* aorta and ascending portion of the aortic arch tends *to extend forwards and outwards, producing a swelling which may be seen and* felt pulsating in the right second and third intercostal spaces. It is also dull to percussion and often tender on pressure. It sometimes erodes the ribs and sternum Pain is usually present and may be the result of bony erosion or be directly due to aortitis. If the aortitis has spread to produce stenosis of the coronary orifices the pain takes on the features of cardiac ischaemia. Similarly, if in the course of its extension it has produced dilatation of the aortic ring, evidence of aortic incompetence is present. If in the absence of hypertension or gross atherosclerosis the aortic second sound is accentuated, aortic aneurysm should be suspected The aneurysm may press on the superior vena cava producing venous distension, cyanosis of the head and neck, and oedema of the arms Rarely it ruptures into the superior vena cava or into the pulmonary artery, in either case it introduces the features of arterio-venous aneurysm, and in the second circumstance prominent dilatation of the pulmonary artery and distension of the right auricle and ventricle is seen on cardioscopy. Pressure on the innominate artery will produce a small brachial and radial pulse, but this sign may also be the outcome of aortitis extending to involve the innominate artery (Fig. 175), giving rise to an aneurysm, which may occasionally be felt above the right clavicle, again, the orifice of the innominate artery may become stenosed from aortitis, or thrombosis may occur within an innominate aneurysm, in both instances the pulse on the right side is again small. If the pulses differ greatly the blood pressure is diminished on the side of the smaller pulse A difference of over 30 mm of Hg between the two sides suggests the presence of aortic aneurysm. The aneurysm may also press on the right bronchus causing a cough, reduced air-entry to the upper zones of the right lung, and occasionally bronchiectasis. Aneurysm of the ascending aorta may rupture into the pleura or into the tissues of the chest wall and even externally.

An aneurysm of the transverse and descending portions of the *aortic arch* is deeply situated and is not visible as a swelling except when examined radiologically. Its presence is shown by symptoms which arise from pressure on adjoining structures. The trachea may be deviated prominently to the right and its lumen constricted (Fig 176) The left bronchus may similarly be pressed upon and give rise to a characteristic ringing or brassy cough, inspiratory and expiratory stridor, dyspnoea, diminished air-entry into the left lung, and sputum and haemoptysis from bronchiectasis. If the cricoid cartilage is held between the thumb and forefinger with the patient erect and the chin elevated, a sensation of tugging (tracheal tug) has sometimes been felt at each aortic pulsation, but this is seldom

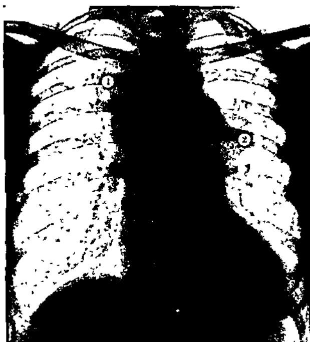


FIG. 175—Innominate aneurysm (1) Another aneurysm involves the descending aorta (2) Absence of the radial pulse had been observed in this patient, a female aged 65 years, for 15 years



FIG 176—Deviation of the trachea (1) to the right by an aorta (2) elongated by hypertension and dilated from aortitis, seen in the left oblique position Aneurysmal dilatation of ascending aorta (3) and descending aorta (4) Enlargement of the left ventricle (5)

a helpful or reliable sign. The left recurrent laryngeal nerve is often involved as it courses round the aortic arch to pass behind it. The abductor fibres of the nerve succumb to the effects of pressure before the adductor fibres, and the vocal cord is at first in the position of adduction. At this stage there is not alteration of the voice during either phonation or respiration, for in each circumstance the right vocal cord moves up to the already adducted left cord. Laryngoscopy, however, will disclose the presence of this early change before the onset of symptoms. As the pressure increases, the adductor fibres become affected and the left



FIG 177—Aneurysm of the lower part of the descending thoracic aorta (1) behind the heart jerks the heart forward during systole. Aneurysm of the arch (2) and upper part of descending aorta (3) as well Dilatation of ascending aorta (4)

vocal cord remains motionless in the cadaveric position half way between full adduction and abduction. The voice then becomes hoarse and often provides the presenting symptom of aneurysm of the aortic arch. Pressure on the sympathetic nerve first causes irritation and later paralysis of the cervical sympathetic fibres; when paralysis has taken place the pupil on the corresponding side is smaller, and the eyeball is sunken in the orbit (enophthalmos); this constitutes Horner's syndrome. Unequal pupils in a case of aortic aneurysm may occur independently of sympathetic involvement, thus Argyll Robertson pupils may be present, or enlargement of one or both pupils may result from

diminished pressure in the carotid arteries and in the spiral arteries to the iris from pressure of the aneurysm on the main arteries in the neck. Although aneurysm of the aortic arch causes considerable displacement of the oesophagus to the right it seldom produces dysphagia, such displacement of the barium-filled oesophagus is helpful in radiological diagnosis.

In the absence of aortitis in the beginning of the aorta, aneurysm of the *descending thoracic aorta* may remain undiscovered for some years until some symptom, such as pain in the chest, leads to radiological examination. Occasionally a large aneurysm of the lower part of the descending thoracic aorta forces the heart forwards against the left chest during systole, producing the **thoracic jerk** (Fig. 177). Sometimes the aneurysm presses against the bodies of the thoracic vertebrae and causes bony erosion.

In the absence of radiology the diagnosis of a thoracic aneurysm will usually remain in doubt unless such pressure symptoms as typify an aneurysm of the aortic arch are present The added evidence of aortic incompetence should lead to a radiological investigation, and since aortitis is a frequent cause of the pain of cardiac ischaemia each patient the subject of this symptom should be similarly examined It has already been pointed out that in the radiological diagnosis of early aortitis care should be taken to exclude hypertension and medial degeneration as causes of a disturbed aortic outline The differentiation of the more gross deformity due to aneurysmal dilatation from intrathoracic neoplasm is seldom in doubt when the swelling has been viewed in the anterior, left oblique, and right oblique positions. The advantages of gradual rotation of the patient during radioscopy are well illustrated in the serial teleradiograms shown in Fig 178. Further help is obtained from the direction in which the barium-filled oesophagus is displaced by the vascular swelling in each of the three orthodox views (Fig 179) An aneurysm of the ascending aorta and ascending portion of the aortic arch does not displace the oesophagus. If situated in the transverse or descending portion of the arch it displaces the oesophagus to the right (Figs 180 and 181) when viewed in all three positions, but only slightly to the right in the left oblique view. The aortic arch impression is elongated proportionately to the increased diameter of the aorta If situated in the upper portion of the descending thoracic aorta the barium stream is again displaced to the right in all three positions (Fig 182), but only slightly in the right oblique view. Aneurysm of the middle portion of the descending thoracic aorta displaces the oesophagus slightly to the right in the anterior view, to the right in the left oblique and slightly to the left in the right oblique view (Fig 183). Inspection of the barium-filled oesophagus gains greatest importance in discovering an aneurysm of the lowest portion of the descending thoracic aorta The aneurysm usually displaces the oesophagus to the left in the anterior and right oblique views (Fig 184), and slightly to the right in the left oblique view (Fig. 185)

Aortitis involving the abdominal aorta is a much less common finding than that involving the thoracic aorta Similarly, aneurysmal dilatation in this situation is comparatively uncommon When of moderate dimensions it can be felt as a pulsatile abdominal swelling situated in the course of the aorta It may be possible to determine that the pulsation is expansible in character. It is difficult to demonstrate the vascular nature of the swelling in this position by means of



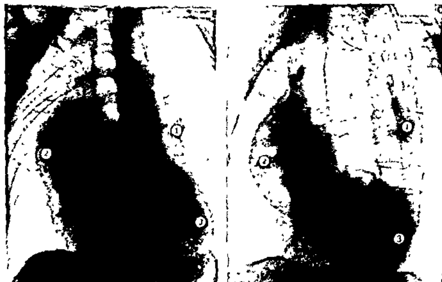


FIG 178.—Aortitis, aortic incompetence, and aortic aneurysm Slight rotation to the right in (B) shows dilatation of the descending aorta (1) to a better advantage The orthodox left oblique view in (C) displays best the aneurysm of the ascending aorta (2) Enlargement of the left ventricle (3) The wall of the aneurysm is calcified

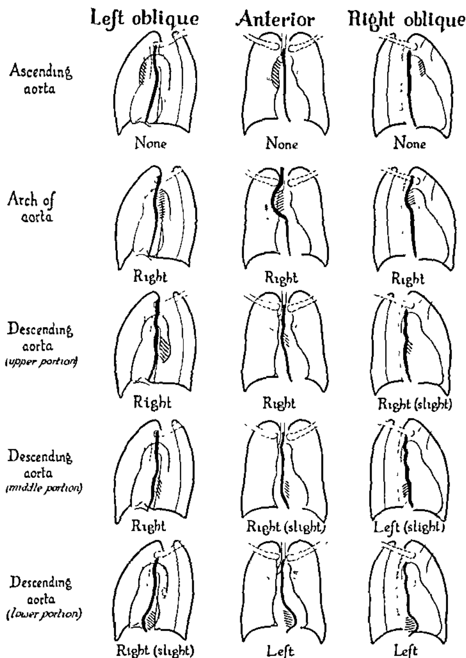


FIG. 179—Depicting the oesophageal impressions in the three orthodox positions from aneurysm situated in different portions of the thoracic aorta. The direction of the oesophageal displacement is indicated in each view and nominated for each aneurysm



FIG 180—Aneurysm of the aortic arch (1) The natural aortic arch impression is greatly exaggerated in depth and in length Male aged 50 years (Post mortem control)



FIG 181—Aneurysm of the aortic arch (1) in the left oblique view The aortic arch impression is deep and greatly elongated The left bronchus impression (2) is consequently much more abrupt Male aged 50 years. (Post-mortem control)



Fig 182.—Aneurysm of the upper part of the descending aorta (1) displacing the oesophagus to the right in the left oblique view, and making the left bronchus impression (2) less prominent. Male aged 62 years



Fig 183—Aneurysm of the middle portion of the descending thoracic aorta (1) displacing the oesophagus slightly to the left in the right oblique view. The aortic arch impression (2) is also elongated in this case from aortitis which has produced dilatation of the arch. Male aged 62 years



FIG. 184—Aneurysm of the lowest portion of the descending thoracic aorta (1) displacing the oesophagus to the left in the right oblique position In a less exaggerated form this might resemble the "cascade" curve from the elongated aorta of atheroma Male aged 47 years



FIG 185—Aneurysm of the lower part of the descending thoracic aorta (1) displacing the oesophagus slightly to the right in the left oblique position Normal aortic arch impression (2), left bronchus impression (3), and left ventricle (4)

radiography unless its wall is calcified (Fig. 186) The presence of aortitis affecting the thoracic aorta and of other clinical evidence of syphilis, together with a positive Wassermann reaction, will render the diagnosis of abdominal aneurysm a probable one, provided that it has been possible to exclude a tumour connected with any of the abdominal viscera.

COURSE AND PROGNOSIS

Although patients with syphilitic aortic aneurysm often live to an old age, the liability to rupture when the condition is long established always renders the prognosis uncertain Rupture may take place into the pericardium, pleura, oesophagus, bronchus, lung tissue, or anterior chest wall, and prove fatal. The



FIG 186—Two abdominal aortic aneurysms (indicated by arrows) with calcification of their walls in a male aged 48 years who also showed thoracic aortic aneurysms

development of pressure effects such as bronchiectasis, or the presence of aortic incompetence precipitating heart failure, adds to the seriousness of the condition The persistence of pain due to cardiac ischaemia after a period of treatment with anti-syphilitic remedies should be viewed unfavourably, and when involvement of the coronary orifices progresses to produce cardiac infarction the outlook is immediately very grave. Although anti-syphilitic remedies will often relieve the pain of aortitis and prevent rapid extension if applied early, they cannot, of course, bring about retrogression and diminution in the size of an aneurysm.

TREATMENT

There is little to recommend in the general treatment of patients with aortitis. They may be permitted to follow their usual vocation provided that it does not

involve undue exertion. The benefit obtained from restricted diet is found in the prevention of obesity in a patient when limitation of activities has been advised. Much has been written concerning the best form of medicinal treatment in patients with syphilitic aortitis The following therapeutic measures have been advocated · mercury and iodide by mouth over a long period ; weekly subcutaneous injections of 0·2 gramme of bismuth for three months or until signs of intolerance develop , ten deep injections of arsenic dispensed as follows. five weekly injections of 0 3 gramme, and after three weeks' rest a further course of five weekly injections. Although the use of arsenical preparations in aortitis has been opposed, when they have been prescribed under proper supervision I have not met with any untoward

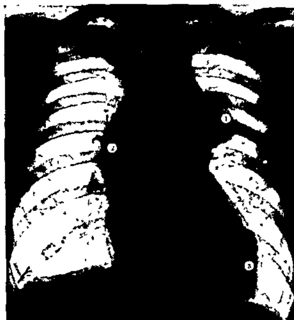


Fig 187 —Aneurysm (1) of commencement of descending aorta grew rapidly under observation during two months, and benefited greatly from wiring Dilatation of ascending aorta (2) Enlargement of the left ventricle (3) from aortic incompetence.

symptoms It is impossible to assess the value of a drug in the treatment of aortitis by attempting to estimate by radioscopy the progression or otherwise of a portion of aorta bulging as the result of aortitis The effects of a drug are best evaluated in those patients in whom pain of cardiac ischaemia is a feature. If pain is relieved it is right to assume that the drug is benefiting the underlying aortitis Thus, in a series of controlled cases in which these various anti-syphilitic remedies were submitted to clinical trial, mercury and iodide by mouth proved to be the best form of treatment to adopt Mercury may be prescribed as 1 drachm of liquor hydrarg. perchlor., and iodide as 5 grains of potassium iodide three times daily for upwards of twelve months. The advantages of larger doses of iodide are not proven. If heart failure appears in the cases in which aortic incompetence is present, adequate rest should be provided and mercurial diuretics

and digitalis prescribed. If the pain of cardiac ischaemia is a feature, tablets of glyceryl trinitrate, when chewed, will relieve the attacks, and similarly will prevent the onset of expected attacks when taken beforehand The surgical treatment of aortic aneurysm by the passage of a wire into the sac, is occasionally carried out, but without much benefit in the majority of cases. It is best reserved for those cases in which the aneurysm is very localized, presenting to the right or to the left of the sternum, and has a narrow neck, and is extending quickly (Fig. 187).

CHAPTER 9

CARDIAC PAIN

THE TERMINOLOGY applied to pain arising from disease of the heart has to be revised This revision has become necessary partly because cardiac infarction has received recognition as a separate clinical entity, and partly because there has been included under the general term *angina pectoris*, pain in the chest which is not of cardiac origin. To gain simplicity and preciseness in the diagnosis of heart pain one might be helped by discarding the term angina pectoris and substituting instead a nomenclature implying the cause of the clinical syndrome. Thus, cardiac pain might be held to be due to cardiac ischaemia or cardiac infarction, and pain in the chest not caused by one of these two conditions should not bear reference which might infer an association with the heart.

Before discussing the two varieties of heart pain it is necessary to discuss its differential diagnosis from another kind of chest pain, namely the so-called *inframammary pain* It is common in all nervous subjects at all ages Although made worse at times by exercise, its production is not dependent on physical effort and it commonly lasts while at rest. In young subjects it has been counted as part of the condition named " effort syndrome ", and it is common in nervous subjects with mitral stenosis It is of greatest significance in older patients for in them the diagnosis is most likely to miscarry , such patients, more commonly females than males, are nervous and often show moderate hypertension , the pain may be associated with tenderness and felt in the left subscapular area as well

TABLE V

A list of terms commonly employed by patients to describe pain of cardiac or non-cardiac origin

Cardiac pain	*Non-cardiac pain*
Indigestion	Shooting
Dull	Sharp
Aching	Throbbing
Tightness	Stabbing
Pressure	Piercing
Constricting	Knife-like
Band-like	Niggling
Vice-like	Sticking
Heavy	Stinging
Crushing	Pricking
Gnawing	Fluttering
Boring	Cutting
Choking	Twinge
Gripping	Knocking
Burning	Stitch

True heart pain is usually described by the patient in undemonstrative terms (*see* Table V) intended to underestimate the symptom The nervous patient with

functional pain, on the other hand, is eager to impress others of the reality and even the severity of his symptom, and such an attitude inevitably leads to exaggeration.

The two varieties of heart pain, namely, cardiac ischaemia and cardiac infarction, will now be described.

CARDIAC ISCHAEMIA

Cause of the pain in cardiac ischaemia

Experiments in animals have demonstrated cardiac ischaemia in temporary obstruction of the coronary artery, but the exact mechanism of the painful paroxysm in man is difficult to substantiate The ischaemia may be precipitated

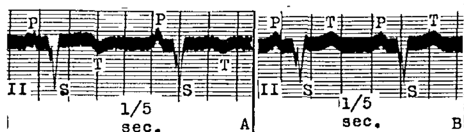


FIG 188.—Inversion of the T wave from coronary occlusion (A), corrected by inhalation of amyl nitrite (B)

eventually by a spasm of the coronary artery. This belief gains support from finding that the inverted T wave of the electrocardiogram, consequent on ischaemia, can be corrected by a vasodilator such as nitrite (Fig 188).

Within the clinical *syndrome of cardiac ischaemia it is possible to recognize* two distinct groups The first is due to coronary disease, and the second to coronary anaemia or insufficiency of the coronary circulation from a depleted blood supply due to causes other than disease of the coronary arteries The liability of the first group to develop the major complication of cardiac infarction gives to it a peculiar importance which is not shared by the second group in which this complication never appears.

CARDIAC ISCHAEMIA FROM CORONARY DISEASE

Atheroma of the coronary artery is the commonest cause of cardiac ischaemia and accounts for about 90 per cent of cases Stenosis of the coronary orifice by *syphilitic aortitis* is also a cause and is responsible for about 10 per cent. Ischaemia of the myocardium from these two sources may lead to patchy areas of degeneration, atrophy and slight loss of muscle fibres, and replacement fibrosis, but the major *catastrophe of cardiac infarction does not take place unless occlusion* of a main branch of the coronary artery is either severe, or permanent from thrombosis.

SYMPTOMS

Although cardiac ischaemia is occasionally met with in younger subjects it is usually found after the age of 40 years. The condition is commoner in males than in females. It is characterized by attacks of pain in the chest variably described by

the patient as a dull heavy ache or pain, a sense of tightness, indigestion, constriction or pressure. The severity of the attacks varies, but seldom are they severe enough to be accompanied by a fear of impending death (angor animi). The majority of patients are not demonstrative during the attacks, and are non-complaining in the expectation that rest will soon bring about relief from the pain. They prefer to remain still, but if interrogated they will converse quietly. If the opportunity presents they will sit down, but otherwise they will remain standing or will even continue to walk slowly if the attack is not severe or the journey not uphill. The pain in cardiac ischaemia is felt behind the sternum in its middle, upper or lower third, although sometimes it may be localized in the left side of the chest and even in the back between the shoulder blades It may spread to the left shoulder and down the left arm as far as the elbow, wrist or fingers, to the throat and jaw, to the back, or down the right arm Occasionally the pain starts in the left wrist or in both wrists and spreads into the chest The paroxysm is brought on by exertion, or by emotional disturbances, but only where physical movement would bring on the pain as readily. The nature and extent of the exciting cause will vary among individuals ; thus when the attacks appear frequently the least exertion or even the slightest annoyance may bring on a painful paroxysm. Walking is the most common form of physical effort to precipitate an attack particularly if executed quickly or uphill, or even if proceeding slowly on the flat or against a cold and strong wind. Arm exercises such as sawing, planing or weight-lifting will readily induce attacks, and so will towelling, shaving, stooping, dressing or undressing, in some patients The attack may also occur at night and such a patient is not always troubled by disturbing dreams Exercise taken immediately after a meal particularly renders a patient liable to develop the pain The frequency of attacks of cardiac ischaemia is subject to a natural variation so that a patient who suffers habitually from multiple attacks during the day, may for a period of time experience only few, although indulging in the same amount of exercise as previously. Again, a patient experiencing one or two attacks daily may remain completely free from pain for some time Such periods of natural variation, or even remission, are of great importance when the efficiency of a remedy in preventing attacks is under consideration Although a patient with cardiac ischaemia may often complain of shortness of breath, interrogation will establish that the sensation is one of shallow breathing, a desire to take a deep breath, rather than one of rapid and distressing breathing Cardiac ischaemia is almost entirely subjective in its manifestations. The pulse rate may not change during the paroxysm, but the blood pressure may become slightly raised. Occasionally auricular fibrillation may be present in a patient, and seldom do attacks of cardiac ischaemia disappear with the onset of fibrillation.

DIAGNOSIS

Although cardiac ischaemia does not present any constant objective symptoms, the condition is presumed to be present when a patient over the age of 40 years experiences recurrent attacks of pain spreading across the chest and often radiating into the back or shoulders or down one or both arms and sometimes up to the throat and the lower jaw, and when the attack is induced by physical exertion and is quickly relieved by rest or nitrite. Hypertension or aortic incompetence (usually

syphilitic in this instance) should be sought in any patient suspected of cardiac ischaemia, and the presence of either will give support to the diagnosis, so also will the presence of triple heart rhythm as an expression of heart failure. One condition which is likely to give difficulty in diagnosis is cardiac infarction and the differentiating features of the two common syndromes are best outlined in a table (Table VI).

Even when due regard is paid to the separate features shown in Table VI it is common experience to find that patients with all the clinical characteristics of cardiac ischaemia, show changes in the *electrocardiogram* which denote cardiac infarction The importance of this test in all cases of heart pain thereby receives emphasis. Naturally, a chest lead should be recorded whenever the limb lead cardiogram is equivocal, and even if the limb lead tracing is absolutely normal provided that the clinical history is typical of cardiac ischaemia. Should the T_3 of an otherwise normal cardiogram be inverted, the effect on it of deep inspiration should be tested for if this is a physiological change the respiratory manoeuvre will partly or wholly correct it. Significant deviation (2 mm.) of the S–T segment of the electrocardiogram, especially with depression of the T wave, after exercise or an anoxaemia test, may help to determine the presence of coronary arterial disease in patients with chest pain when the routine cardiogram does not show any obvious abnormality.

COURSE AND PROGNOSIS

The frequency and severity of the attacks, the age and activities of the patient, together with the presence or absence of clinical, cardiographic and cardioscopic evidence of cardio-aortic disease, should all be considered when attempting to assess the prognosis in a patient with cardiac ischaemia Even then it must always remain uncertain since the occurrence of cardiac infarction cannot be predicted. This uncertainty should not make the outlook in cardiac ischaemia invariably ominous, because individual patients may continue to suffer from the attacks for 5, 10 or even more years, and to the patient at least a fair share of reassurance is due, especially when the *electrocardiogram* is normal, in which case coronary disease may not be the cause.

TREATMENT

Although the course of the illness, except in its syphilitic form, cannot be materially altered by any remedy, and the onset of cardiac infarction cannot be prevented or postponed, much can be done to reduce the frequency and severity of the attacks Much is gained from precise instructions concerning daily work and habits. If a patient's occupation entails considerable physical effort, he must forego it and follow one which involves less exertion. Provided that exertion is avoided, indulgence in moderate exercise may be permitted and even encouraged. Rest immediately after meals, although not essential, may reduce the number of attacks. Meals should always be light, and if obesity is present much benefit is anticipated from a reduction of the patient's weight following adherence to a fat-reducing diet. In cardiac ischaemia due to syphilitic aortitis it is possible to direct treatment to the causative lesion. As a generalization it is true that once a patient is subject to attacks of cardiac ischaemia he is always liable to them unless they have arisen in the course

TABLE VI

Comparing the main clinical features of cardiac infarction and cardiac ischaemia.

Features		*Cardiac Infarction*	*Cardiac Ischaemia*
Aetiology		Coronary atheroma (90 per cent) Syphilitic aortitis (9 per cent) Coronary embolism (1 per cent)	Coronary atheroma (90 per cent) Syphilitic aortitis (9 per cent) Coronary anaemia (1 per cent)
Mechanism		Permanent coronary occlusion, infarction	Temporary cardiac ischaemia, recovery without infarction
Pain	Onset	Often at rest	Following exertion or emotional disturbance
	Site	Often in lower one-third of sternum	Commoner in middle one-third of sternum
	Radiation	Usually extensive	Usually less extensive
	Duration	Hours or days	Few minutes
	Relief	Rest and morphine	Rest or nitrite
Restlessness		Common	Absent
Dyspnoea		*Often present*	*Absent*
Vomiting		Not infrequent	Rare
Temperature		Subnormal at onset; later pyrexia for 5–7 days	Normal
Leucocytosis		Usually present	Absent
Sedimentation rate		Raised	Normal
Pulse		Small, often rapid, occasionally irregular	Usually no change or a little rapid
Blood *pressure*		Often considerable fall of systolic *blood pressure; small pulse* pressure	No change or raised
Heart sounds		Distant, often triple rhythm, occasionally pericardial friction sound	Occasionally triple rhythm
Heart failure		Often present	Absent
Electrocardiographic changes		Deviation of R–T segment with T wave inversion	Often changes as in cardiac infarction

of syphilitic aortitis, in a proportion of these cases the painful paroxysms are prevented, at least for a period, by anti-syphilitic remedies, among which mercury and iodide by mouth have given the most consistent satisfactory results When attacks of cardiac ischaemia take place at night the determined use of hypnotics has proved of great value either in reducing the number of attacks or even in preventing them. Phenobarbitone or chloral hydrate in adequate doses has proved as efficient as morphine in this connexion. Whenever the attacks are of recent onset and hypertension is a feature, a resting period of six weeks in bed has occasionally reduced both the frequency and severity of the attacks. Many are the medicinal remedies advocated in the continuous treatment of cardiac ischaemia, but *out of fifteen active drugs possessing a reputation in this connexion none proved,* in a series of patients, to be much more efficacious than a simple placebo mixture (Fig. 189). Again, many remedies have been recommended to relieve the actual attack of cardiac ischaemia, and when the comparative value of eleven such preparations was estimated, glyceryl trinitrate (trinitrin) tablets, when chewed, proved to be by far the best (Fig 190) Similarly, this tablet proved invaluable when used to prevent the onset of expected attacks, and prophylactic treatment of this nature should be urged in patients presenting this condition (Figs. 191 and 192); *indeed, therein lies the most valuable form of treatment for a patient with* cardiac ischaemia

Instructions on the use of trinitrin tablets.—The patient should be told to keep a two-months' supply of tablets in a stoppered bottle stored in a dry, temperate and darkened cupboard A few tablets are carried in a suitable box, readily accessible for use when needed The patient must be reassured about the safety of the tablets and their short-lived action. They should be chewed as the drug is absorbed quickly by the buccal mucous membrane, and hardly at all from the stomach (Fig 193) A tablet ($\frac{1}{100}$ gr) should be chewed whenever the painful paroxysm has set in, but emphasis must be laid on the importance of trying to prevent the onset of an attack by chewing half a tablet while undergoing that form of exercise which customarily induces an attack It is advisable to forewarn the patient at the first interview that a sensation of fulness in the head is a natural outcome of ingestion of trinitrin, this precaution obviates the apprehension which might otherwise arise when trinitrin is taken for the first time

CARDIAC ISCHAEMIA FROM CORONARY ANAEMIA

The symptoms in this group are indistinguishable from those in cardiac ischaemia due to coronary disease. The prognosis, however, is quite different because of the liability of the first group to cardiac infarction, a complication which would only be fortuitous in the second group The importance of this kind of cardiac ischaemia lies with differential diagnosis, for it should be distinguished from the more serious kind. An impoverished coronary circulation in the absence of coronary arterial disease is found in the following clinical states

The pain of cardiac ischaemia is among the commoner symptoms of a moderately severe *systemic anaemia* To be aware of this is to examine the conjunctiva in patients complaining of this pain, especially if they are women. Treatment for the anaemia dispels the pain and there is no more need for trinitrin.

The symptom is sometimes met with in *mitral stenosis* and the addition of aortic incompetence does not determine its presence in a given case. A mass thrombus in the left auricle has been found sometimes at necropsy in a case of mitral stenosis that presented cardiac ischaemia during life, but it is unlikely that such a finding is invariable in patients with this symptom.

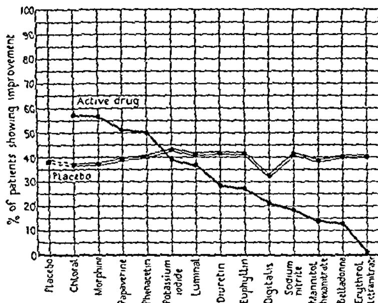


FIG 189.—Diagram showing comparative improvement in patients with angina pectoris from active drugs and a placebo Continuous line indicates improvement from active drugs Double line indicates improvement from placebo in the same patients Interrupted line indicates improvement in a standard series of patients treated with placebo

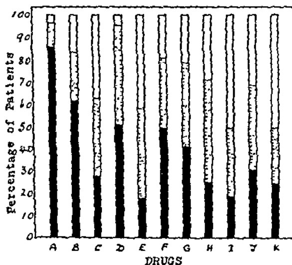


FIG 190.—Benefit from remedies used in the immediate treatment of attacks of cardiac ischaemia in 122 patients The black columns indicate *great relief*, the shaded columns indicate *moderate relief*, and the unshaded columns indicate *no relief*. (A) Trinitrin tablets (B) One per cent liquor trinitrini. (C) Oily solution of trinitrin (D) Natirose dragees (Nativelle). (E) Nitrolingual capsules (Pohl) (F) Trinitrin caféinee (Dubois) (G) Amyl nitrite (H) Sodium nitrite. (I) Brandy (J) Chloroform (K) Carminatives

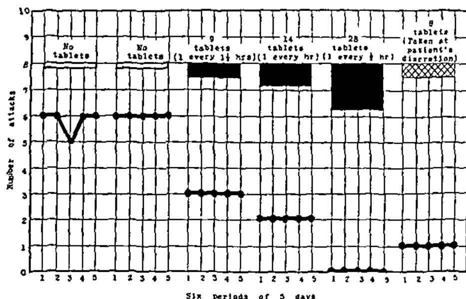


FIG 191 —Illustrating the effect of glyceryl trinitrate (trinitrin) tablets on the incidence of attacks of cardiac ischaemia in a male, aged 59 years. The unshaded columns represent two control periods of 5 days when glyceryl trinitrate was not used prophylactically. The black columns represent three periods when patient took 9, 14, and 28 tablets (gr $\frac{1}{100}$ each) daily at intervals of 1½, 1, and ½ hour respectively The shaded column indicates a period when the patient took 8 tablets daily, using them at his own discretion for the prevention of expected attacks The observation was repeated so that natural variation in the incidence of attacks can be discounted

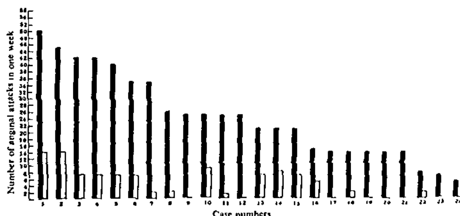


FIG 192 —Illustrating the effect of the prophylactic use of glyceryl trinitrate (trinitrin) tablets on the incidence of attacks of cardiac ischaemia in 24 patients The black columns represent the number of attacks in each patient when tablets were not used to prevent them The unshaded columns indicate the number of attacks when each patient took glyceryl trinitrate tablets at his own discretion to prevent the onset of expected pain The attacks are markedly diminished in all, while 8 remained free from attacks following the prophylactic use of the tablets

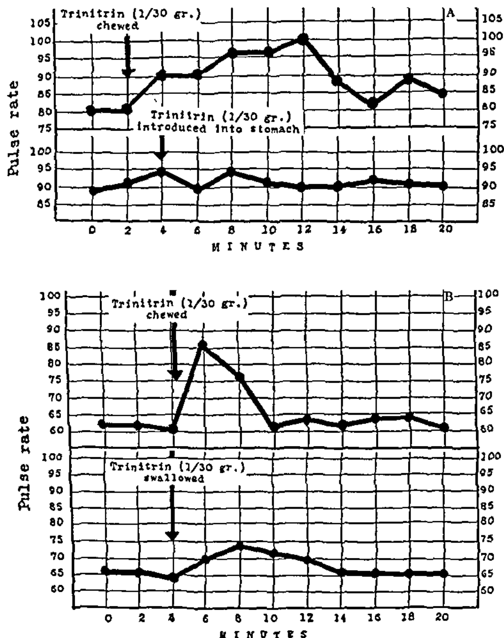


FIG 193—In (A) are seen the effects of glyceryl trinitrate (trinitrin) on the pulse rate in a male, aged 71 years, in whom gastrostomy had been performed for carcinomatous obstruction of the oesophagus, the first tracing was recorded after chewing trinitrin, and the second following direct introduction of trinitrin into the stomach The effects of glyceryl trinitrate (trinitrin) on the pulse rate in a male, aged 23 years, when chewed and when swallowed, are shown in (B)

The interpretation of cardiac ischaemia in a patient with *aortic stenosis* will often give rise to difficulty as to whether the cause of the pain is coronary disease or an impoverished coronary circulation consequent on the aortic stenosis. That this mechanism of coronary anaemia takes place is shown by the absence of coronary arterial disease in cases of aortic stenosis examined at necropsy. The electrocardiogram can seldom help, because it so frequently shows abnormal changes from aortic stenosis which are not very unlike those in cardiac infarction.

During *paroxysmal tachycardia* retrosternal pain is a prominent symptom in some patients It differs from the usual pain of cardiac ischaemia in that it lasts longer, and often for as long as the attack does. It has been shown that the presence of pain in such cases is not any index of the state of the coronary arteries.

Painful attacks occur during the tachycardia of *thyroid toxaemia*, but they are rare.

Sometimes the pain of cardiac ischaemia has been induced by injections of *adrenaline* or by breathing air of a *lowered oxygen* content, and use of this has been made to test the presence of coronary artery disease. *Insulin* is also reported to have induced chest pain.

Another condition, which presents pain on exertion with the same distribution in the chest and arms, appears to have a dyspeptic source, and an investigation of this is proceeding.

CARDIAC INFARCTION

Although a clinical diagnosis of cardiac infarction was made on occasion by isolated observers as long ago as half a century, its recognition as a separate clinical entity has only become general within recent years.

AETIOLOGY

Coronary atheroma supplies the commonest cause of cardiac infarction, and is operative in about 90 per cent of cases. Syphilitic *aortitis* implicating the coronary orifices is the cause in about 9 per cent, and coronary *embolism* resulting from valvular vegetations or thrombi in dilated heart cavities in the remaining 1 per cent. At necropsy, in cases in which coronary atheroma provides the cause of infarction, thrombosis is often absent and atheromatous constriction by itself may be the cause, especially when atheroma also involves the collateral circulation. In those cases in which stenosis of the coronary orifice by syphilitic aortitis is the cause, coronary thrombosis is seldom present. A common site for coronary thrombosis from atheroma is the anterior descending branch of the left coronary artery in the first 3 cm. of its course. The area of infarction may be very small or it may be so extensive as to involve almost the whole of the left ventricle. The site of the infarct depends on the particular artery affected and the position of the thrombosis within the artery. Occlusion of the *anterior descending branch of the left coronary artery* will usually lead to infarction of the apex, anterior wall of the left ventricle, and the anterior part of the interventricular septum. Occlusion of the *circumflex branch of the left coronary artery* is likely to produce infarction of the lateral and posterior walls of the left ventricle. Occlusion

of the *right coronary artery* will give rise to infarction of the posterior part of the interventricular septum and posterior walls of both ventricles. If the affected artery assumes an abnormal anatomical course the distribution of the infarcted areas from thrombosis will vary accordingly. When infarction is recent its limits may be difficult to define, but a section through the heart shows pallor and bulging of the cut surface especially towards its endocardial aspect. Older infarction appears as a yellow or grey area often punctuated by haemorrhages and sharply defined by a linear haemorrhagic border. Still older infarction is marked by a shrunken grey or white area of muscle which is thin and occasionally bulging on its pericardial aspect from an aneurysm.

SYMPTOMS AND DIAGNOSIS

Cardiac infarction, commoner in the male than in the female, usually occurs after the age of 40 years, although it sometimes takes place in subjects under 30 years of age. It may cause sudden death, but usually its presence is signalled by pain in the chest which may set in while the patient is at rest and commonly during the early hours of the morning. The pain is situated behind the middle or lower part of the sternum, and its distribution, as a rule, is more extensive than in cardiac ischaemia; thus it may spread to the epigastrium, to the back, to the shoulders and down one or both arms, and to the throat and lower jaw. The severity of the pain often causes restlessness and a patient seeks in vain a position of ease. Breathlessness is a common symptom when infarction is extensive, and this may be so severe as to overshadow the symptom of pain. Vomiting may occur, and a state of shock, with pallor and sweating, is a common symptom. Unconsciousness may happen, especially in the presence of an arrhythmia.

In a number of patients, in whom the electrocardiograms prove cardiac infarction and in whom the symptom of pain is recent, this characteristic story is absent, the attack of pain is brief, much less severe, and simulates closely the pain characteristic of cardiac ischaemia.

At the onset of the severe attack the temperature is subnormal and this is followed by a moderate and irregular pyrexia lasting about five days, leucocytosis and a raised sedimentation rate are associated with this pyrexial phase. The pulse is small and often a little rapid. Although usually regular it may sometimes be irregular from auricular fibrillation, paroxysmal tachycardia, heart block or pulsus alternans The systolic blood pressure is lowered producing a small pulse pressure, for example, 95/80. If hypertension has been a feature, the systolic blood pressure may have fallen to a normal figure, but the diastolic blood pressure in these cases may often remain raised, permitting the inference that a state of hypertension had been present before the onset of the attack of cardiac infarction. The recovery in the blood pressure value may take several months, and often it may never take place. The heart sounds are usually distant and sometimes a pericardial friction sound is heard Evidence of heart failure is frequently an early event in the illness, so that crepitations are heard over the lung bases, sometimes moist sounds are so conspicuous and generalized as to suggest the diagnosis of acute pulmonary oedema. Tender distension of the liver and slight pitting oedema of the ankles may occur early, but frank signs of failure are usually a later manifestation. In this instance hilar congestion determined by cardioscopy precedes other clinical signs of heart failure, although when hypertension is present, and it is a common association in

cases with failure, triple rhythm is often heard. Triple rhythm, from addition of either the fourth or third heart sound, may be present in the absence of hypertension.

In the early phase of cardiac infarction the *electrocardiogram* characteristically

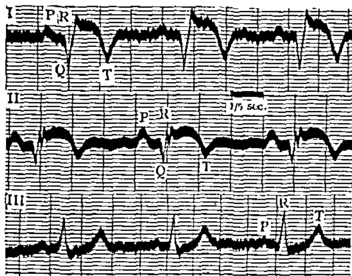


FIG 194.—Cardiac infarction of three days' duration. Prominent R–T deviation, inversion of T wave, and deep Q wave, in leads I and II

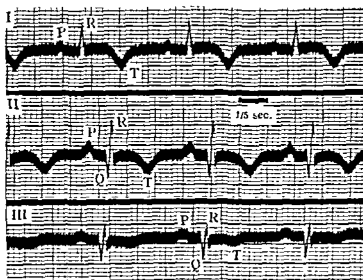


FIG. 195.—Anterior cardiac infarction (T_1 type). Inversion of the T wave in leads I and II

shows deviation of the R–T segment (Fig. 194) followed by inversion of the T wave and deep Q waves in leads I and II (T_1 type of Parkinson and Bedford)

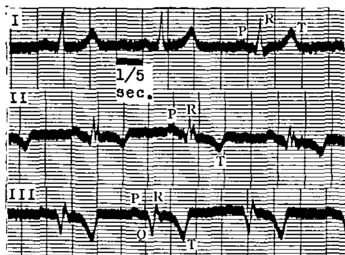


FIG 196—Posterior cardiac infarction (T_3 type) Inversion of the T wave in leads II and III, with deep Q_3

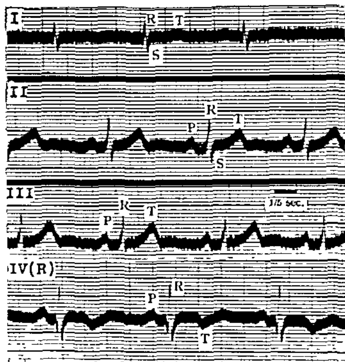


FIG 197—Anterior cardiac infarction The T wave in lead I is upright although low, but inversion of the T in lead IVR confirms the presence of infarction

(Fig 195) or in leads III and II (T_3 type) (Fig. 196) The T_1 type of curve indicates infarction of the anterior part of the heart, and the T_3 type infarction of the posterior part. Deviation of the R–T segment may disappear in the course of a few days, but the associated inversion of the T wave lasts, at least in

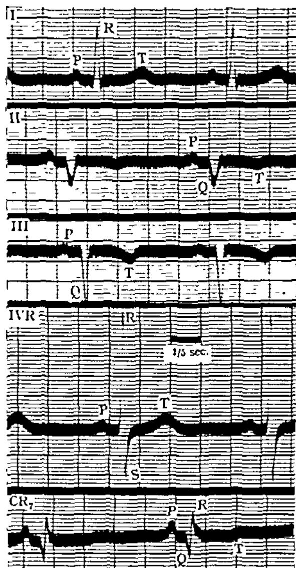


FIG. 198 —Posterior cardiac infarction. The T wave is inverted in lead III, and less in II and CR_7. The Q wave is prominent in leads II and III.

some leads If the changes in the limb lead electrocardiogram are in doubt, then the chest leads CR_1, CR_4 and CR_7 should be taken, for with anterior cardiac infarction the T wave, as a rule, is more obviously inverted in lead CR_4 (Fig 197), and with posterior infarction, the T in CR_7 is either inverted, flat, or low (Fig. 198)

The cardiogram (understood to include chest leads) of cardiac infarction seldom recovers altogether; should recovery of the tracing take place in a suspected case of infarction the explanation may rest with recent paroxysmal tachycardia or pericarditis. In anterior infarction T_2 frequently becomes normal in a short time;

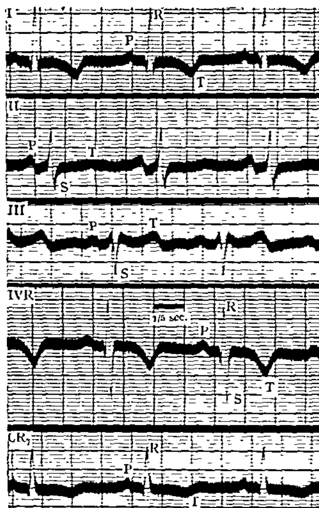


Fig 199.—Anterior cardiac infarction in hypertension Left axis deviation The T wave is inverted in CR_7, but less than in leads I and IVR

T_1 seldom recovers wholly, and still less frequently does the T in CR_7. In posterior infarction the inverted T_3 never returns to normal; T in CR_7 may recover partially, but T_2 seldom resumes its previous upright form. Occasionally inversion of the T wave in leads I and CR_7 is a cardiographic pattern in some cases of infarction, antero-lateral in its distribution.

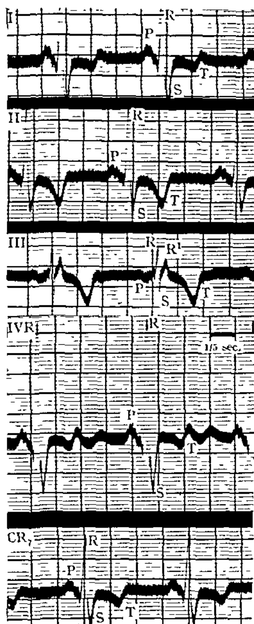


FIG 200—Aortic incompetence The T wave inversion is greater in CR_7 than in IVR The failure of deep inspiration to correct the inversion of the T wave in lead III in posterior cardiac infarction gives help in the cardiographic diagnosis of this condition

The differential cardiographic diagnosis of cardiac infarction is difficult in the presence of hypertension and also when the tracing shows bundle branch block In general it might be said that when T_1 is inverted, if inversion of T in CR_7 is less than in IVR (Fig. 199), it is more likely to be anterior infarction than left ventricular preponderance from hypertension or aortic valvular disease (Fig 200) Inversion of T_2 and T_3 of posterior infarction is told from right ventricular preponderance by a deep S_1, upright T in CR_7, and an inverted T in CR_1, in the latter condition.

COURSE AND PROGNOSIS

A patient with cardiac infarction may succumb suddenly to the first or succeeding attack. Even when the initial phase of the illness is passed, heart failure may follow and the outcome remains uncertain In the more favourable group, the patient emerges from the attack of cardiac infarction without evidence of heart failure and remains free from symptoms apart from occasional slight discomfort in the chest patients in this group have often resumed their usual activities on conclusion of the painful paroxysm, and recovery has been uneventful, taking place without a period of rest. Again, when the first attack has subsided a patient may regain his usual health and remain symptom-free for several months or even years, but usually attacks of cardiac ischaemia set in, and cardiac infarction recurs sooner or later. The prognosis, therefore, must always be uncertain at the commencement of the attack and it must remain indeterminate even in those patients in whom the painful phase of the illness passes after a few hours It is always serious when frank signs of heart failure have appeared, or when the pain persists for a long time, or when the blood pressure remains consistently low Two other complications may take place, namely, embolism, commonly cerebral, pulmonary or systemic, from intracardiac thrombosis, and later, cardiac aneurysm formed during the healing of the infarct.

TREATMENT

The first necessity in the treatment of cardiac infarction is the relief of pain, and morphine must be dispensed determinedly until this end is gained. Sometimes ¼ grain of morphine suffices, but often this will have to be repeated on one or more occasions. The outlook is necessarily grave in those requiring repeated injections of morphine, nevertheless, the procedure should be followed until relief from pain is effected. If heart failure sets in, digitalis and mercurial diuretics need to be given. In the absence of pain and failure, half a grain of phenobarbitone twice or three times a day may be all that is needed in the way of medicinal treatment. Atropine and quinidine have been recommended to prevent shock and ventricular fibrillation, but they are of doubtful value. Absolute rest in bed for a period of six weeks must be enforced in spite of protestations which may come from a patient freed from the initial paroxysm of pain, and convalescence must be a gradual one. That patients who disregard these instructions to take adequate rest can survive and even make good progress is admitted, but the fatal accidents which have overtaken so large a number of patients who have deliberately ignored the instructions to rest, justify the insistence that a patient with cardiac infarction must adhere strictly to this regimen during a time when the infarct is healing, or an intracardiac thrombus, if formed,

is becoming firmly adherent to the endocardial surface. The rest should be as complete as possible and *the patient should neither wash nor shave himself.* Once a day he may be permitted to use the commode or visit the lavatory if it adjoins the bedroom, but otherwise he should use the urine bottle in bed. Such instructions to the patient should be clearly specified and the regimen should not be relaxed during the whole of the resting period. A month's holiday should then be enjoyed before returning to an occupation which may entail a little healthy exercise but which should not involve exertion. Once convalescence has been reached the patient should be given reassurance and encouraged to increase activity gradually, but a decision as to whether to give dissuasion or encouragement to a patient recovering from cardiac infarction must be taken after assessing the temperament of the



FIG 201.—Cardiac infarction showing calcified layers (B) is an enlargement of the left ventricular border from (A) Arrows point to lines of calcification.

individual. *The foolhardy must be told to forego active physical exertion, whereas* those intimidated by the knowledge that they have had a " heart attack " should be given reassurance and encouragement in order to save them from developing a disturbing neurosis.

CARDIAC ANEURYSM

In practice, cardiac aneurysm may be regarded as invariably resulting from cardiac infarction; bacterial endocarditis, congenital maldevelopment, and trauma are other, but rare, causes. The aneurysm forms in the left ventricle, usually in its anterior wall near the apex. It sometimes takes place within a few months of the first attack of pain.

The blood pressure in cardiac aneurysm is seldom raised and often it is low. The apex is displaced outwards and its pulsation is diffuse; occasionally in a thin, elderly subject, this widespread apical impulse may by itself suggest the

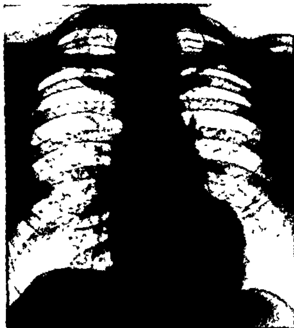


FIG. 202—Cardiac aneurysm following cardiac infarction six months previously The cardiac outline at the bulge indicated by arrow is blurred compared to the outline adjacent to it, this is explained by the exaggerated movement of the wall of the aneurysm during cardiac contraction, and which was a striking feature of cardioscopy

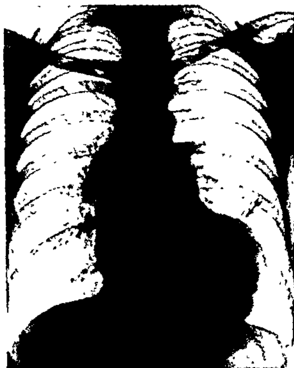


FIG 203—Cardiac aneurysm (indicated by arrow) following cardiac infarction.

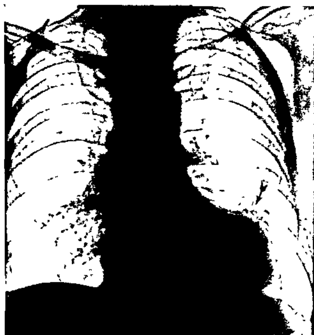


FIG 204—Cardiac aneurysm (indicated by arrow) following cardiac infarction

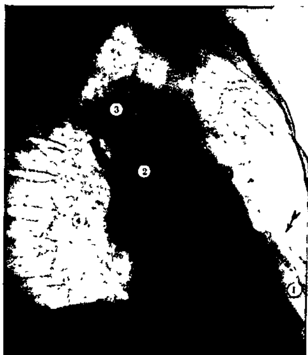


FIG 205—The shelf (indicated by arrow) produced by a cardiac aneurysm as seen in the right oblique position Left ventricle (1) Right pulmonary artery in section (2) Aortic arch (3) Descending aorta (4)

diagnosis A roughish systolic murmur is commonly heard in the mitral area, but unless there is heart failure or bundle branch block associated with a prolonged P–R interval there is no triple rhythm.

The *electrocardiogram*, with rare exceptions, is characteristic of anterior cardiac infarction, showing inversion of the T wave in leads I and CR_1. Exceptionally it shows posterior infarction, sometimes the R–T deviation persists.

Cardioscopy must in the end be the means of proving or disproving the diagnosis of cardiac aneurysm. In rare cases the apex, which shows little pulsation and hardly any bulging, may demonstrate within it a layer of calcification (Fig 201). More commonly a noticeable bulge is plainly seen in the anterior view above the apex (Figs. 202, 203 and 204) and in the right oblique position this appears as a shelf (Fig. 205). The aneurysm following recent infarction often shows vigorous local pulsation, but the older one shows hardly any movement on account of its inner covering of organized thrombus.

Further cardiac infarction, heart failure, or cerebral embolism, is a commoner event than rupture in a patient in whom a diagnosis of cardiac aneurysm has been made. Instances of rupture are more usual during the onset of cardiac infarction and before aneurysm has developed.

CHAPTER 10

HYPERTENSION

DEFINITION

THERE are, as yet, no standard rules which decide when a state of hypertension exists The blood pressure is regarded as raised or not according to the opinion of individual observers on the limits of the normal. It is admittedly difficult to supply a precise definition as to the upper limits of normal blood pressure, but caution is needed in applying the term hypertension when the manometric readings are not raised in any great measure, and in the absence of supporting evidence from pathological changes It needs emphasis that before a diagnosis of *hypertension* is applied to any patient there should be evidence of *cardiovascular hypertrophy*. Particular care should be taken to recognize the change in the blood pressure which might take place from emotional disturbance during the clinical examination, and without any apparent loss of calm on the part of the patient This instability in the blood pressure values during slight emotional upsets is illustrated in Figs 206 and 207 and in Table VII The diastolic blood pressure is not so sensitive to such temporary external influence and is, therefore, a more reliable index of the blood pressure which is standard for a particular patient, but it too may be relatively high according to customary standards in healthy subjects

The following definition is meant to be a guide in the diagnosis of hypertension *The blood pressure is raised when the systolic pressure is* 180 *or over, and/or the diastolic pressure is* 110 *or over, on three consecutive examinations, and in the presence of clinical, radiological and cardiographic evidence of cardiovascular hypertrophy* It should be understood that a state of hypertension may be present even with a normal blood pressure reading, when the previously raised value has been lowered by cardiac infarction, right heart failure, or some other cause.

TERMINOLOGY

Frank used the term *essential* hypertonia to contrast it with *symptomatic*, especially nephrogenous, types of raised blood pressure He also wrote of *primary* hypertension and distinguished it from *secondary* hypertension caused by renal disease. Volhard and Fahr described two kinds of kidney resulting from arterial changes, namely, benign and malignant sclerosis, and referred to the corresponding hypertension as *benign* and *malignant*. Their benign nephrosclerosis corresponded with ischaemic nephritis and their malignant type with the nephritis repens (type 4) of Russell or the contracted red granular kidney of earlier authors. Later, Volhard said that benign or *red* hypertension might change into malignant or *white* hypertension, but that the cause and mechanism of malignant hypertension differed from those of the benign type. Malignant hypertension was caused by a pressor substance in the blood, and this also caused hypertension in other forms of nephritis. Allbutt defined *hyperpiesia* as a hypertension that neither started nor ended with clinical evidence of nephritis Since benign nephrosclerosis may sometimes produce clinical evidence of nephritis, benign hypertension is not synonymous with Allbutt's hyperpiesia.

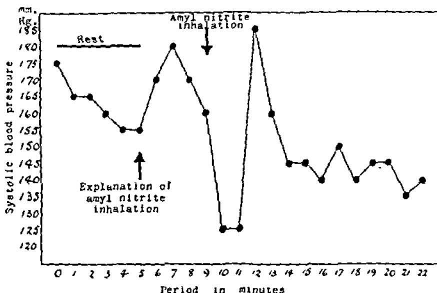


FIG. 206—Changes in blood pressure following rest, emotional disturbance, and amyl nitrite inhalation

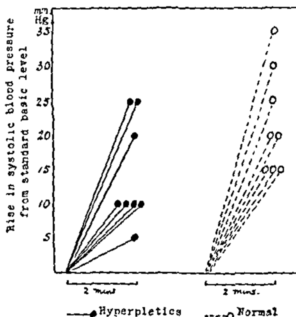


FIG 207—Rise in blood pressure in 8 healthy subjects and in 8 patients with hypertension, following a simple explanation of how to inhale a capsule of amyl nitrite

There is, therefore, clearly a need for unifying our views on the terminology applied to hypertension. Any classification suggested for it should be based on aetiology so far as it is known ; it should recognize that the illness as it progresses might leave one group for another ; it should allow for a change in our views on the different types in accord with newer knowledge as it becomes added,

TABLE VII

The blood pressure in 400 healthy recruits. Examination always included cardioscopy and sometimes electrocardiography. The blood pressure in 40 recruits with early mitral stenosis is shown for comparison

Age	No of cases	*Systolic blood pressure*										*Diastolic blood pressure*						
		Av.	*Less than* 140	140	145	150	155	160	165	170	*More than* 170	*Av*	*Less than* 85	85	90	95	100	105
18	45	150	13	8	1	5	7	4	2	2	3	90	10	16	12	6	1	–
19	57	150	15	6	7	10	4	8	1	3	3	90	8	12	23	9	3	2
20	40	150	7	–	6	11	4	9	1	–	2	90	5	9	11	7	7	1
21	6	150	2	1	–	–	1	–	1	–	1	90	2	1	2	1	–	–
22	8	140	4	1	–	1	–	1	–	–	1	85	3	3	1	1	–	–
23	9	150	1	1	3	2	–	–	1	–	1	90	1	1	3	3	1	–
24	9	150	2	4	–	1	–	–	–	1	1	90	1	1	5	–	1	1
25	14	140	4	4	–	1	4	1	–	–	–	90	3	4	4	2	1	–
26	11	140	4	4	2	–	–	–	1	–	–	90	2	3	3	1	2	–
27	16	145	5	6	–	1	1	–	1	1	1	90	1	5	5	4	–	1
28	12	150	4	1	2	–	–	2	–	1	2	85	3	4	4	–	1	–
29	14	140	7	–	2	1	3	1	–	–	–	90	1	3	7	3	–	–
30	9	150	4	–	1	1	–	–	1	–	2	90	2	1	4	–	–	2
31	13	150	4	–	2	2	1	1	–	2	1	90	2	–	6	3	2	–
32	13	150	4	–	1	1	–	1	2	3	1	90	4	–	3	1	4	1
33	20	145	8	3	1	1	–	2	1	1	3	90	4	4	4	3	5	–
34	19	150	8	2	2	–	–	4	1	–	2	90	4	4	6	3	–	2
35	13	150	4	1	2	–	1	2	–	3	–	90	2	3	4	–	2	2
36	15	150	–	1	2	3	3	2	2	2	–	90	1	3	5	1	5	–
37	11	150	3	1	1	3	–	2	–	–	1	90	1	2	–	5	2	1
38	19	140	9	–	3	1	3	1	–	–	2	90	4	4	6	5	–	–
39	14	145	5	3	–	1	2	1	1	1	–	90	2	3	7	1	1	–
40	13	155	4	1	–	–	–	2	1	3	2	90	1	4	3	5	–	–
	400	150	121	48	38	46	34	44	17	23	29	90	67	90	128	64	38	13
40 cases of mitral stenosis		135	22	4	2	7	2	3	–	–	–	85	9	17	9	5	–	–

and above all else it should be acceptable for discussion at the bedside and in the hearing of the patient ; to this end such a term as *malignant* is unacceptable

PITUITARY HYPERTENSION

Chemical extraction of the posterior lobe of the pituitary gland yields a substance called vasopressin which raises the blood pressure. There is also evidence that the basophil cells in the anterior lobe are concerned, directly or indirectly, with the maintenance of blood pressure. Thus, in Simmonds disease the anterior lobe is destroyed and the blood pressure is consistently low. Again, hypertension is invariably present in the syndrome known as basophilism, when the basophil cells show a peculiar hyaline change which probably represents an increased secretory activity.

ADRENAL HYPERTENSION

The blood pressure is raised by injection of adrenaline extracted from the medulla of the suprarenal bodies and it is also raised by physiological secretion of adrenaline during emotional disturbance Moreover, hypertension is conspicuous in about one-third of the cases with primary tumours of the cells of the suprarenal medulla or other portions of the chromaffin system , in most of these cases, which are rare, the hypertension is paroxysmal. Adrenaline has been found in the tumours and their removal has caused the hypertension to disappear. Hyperplasia, or benign or malignant neoplasm of the suprarenal cortex is occasionally associated with basophilism. As in cases of basophilism which show adenomata in the anterior lobe of the pituitary gland, or in thymic tumours, the basophil cells in adrenal basophilism show the hyaline change. In Addison's disease there is destruction of the suprarenal cortex and also of the medulla, although this may be trivial, and the number of basophil cells in the anterior pituitary gland is greatly reduced. These facts show that there is a close connexion between the suprarenal cortex and the basophil cells of the anterior pituitary gland in their influence on blood pressure.

THYROGENIC HYPERTENSION

Although raised systolic and diastolic blood pressures are sometimes present in thyroid toxaemia, no constant changes are found in the thyroid gland in the common examples of hypertension, nor does thyroidectomy lower the blood pressure in those cases of thyroid toxaemia in which the blood pressure is found to be raised. A commoner change in the blood pressure in thyroid toxaemia is an increase in the pulse pressure from a slight raising of the systolic pressure and a lowering of the diastolic pressure. Hypertension occurring about the time of the menopause in obese women has been attributed to endocrine disturbance, including hypothyroidism, but experimental evidence supporting this view is so far missing

A study of the foregoing conditions leaves no doubt that organs of internal secretion form part of the complicated mechanism which regulates blood pressure. It can also be accepted that hypertension is caused by certain tumours of the suprarenal medulla or other parts of the chromaffin system, certain tumours or hyperplasia of the suprarenal cortex, and some tumours of the thymus. But such causes of hypertension are rare and they are not found in common examples of hypertension. Even if certain morphological changes in the endocrine glands

were found in association with hypertension, such changes might well illustrate the mechanism of the raised blood pressure and not the cause.

HYPERTENSION OF COARCTATION

Congenital stenosis of the aortic arch, dealt with more fully elsewhere, is common enough to cause the adoption of the practice of examining the femoral pulse whenever the brachial blood pressure is found to be raised, especially in young subjects. If the stenosis is proximal to the left subclavian artery the blood pressure in the left brachial artery is low like that of the femoral artery. Aortic incompetence is a commoner event in coarctation hypertension than in the other varieties. It is not unlikely that coarctation hypertension may have a renal cause or effect from ischaemia, for it is becoming apparent that in some cases eradication of the arterial constriction at operation does not always abolish the hypertension.

SIMPLE HYPERTENSION

(*Synonyms*.—Essential hypertension, benign hypertension; primary hypertension; red hypertension, hyperpiesia)

Simple hypertension is the diagnosis given to that clinical state which shows an abnormally raised systolic or diastolic blood pressure, or both, without any cause presenting. Increased peripheral circulatory resistance, neurogenic or humoral in origin, from arteriolar constriction is widespread and not confined to the splanchnic area, but the effect is slight, for the arterioles in simple hypertension may react normally to local and general warming or chilling, and to vasoconstrictor and vasodilator drugs.

PATHOLOGY

The anatomical evidence at necropsy, of a persistently high blood pressure during life is hypertrophy of the heart, especially of the left ventricle, and of the media of the muscular arteries. The changes have been referred to by Turnbull as *cardiovascular hypertrophy*. The intima also hypertrophies in the large elastic arteries of the greater circulation and in the small muscular arteries. When cardiovascular hypertrophy is slight, there is no greater arterial degeneration than in healthy subjects of the same age. As it increases, fatty-hyaline degeneration of some vasa afferentia and recta in the kidneys, and terminal arteries in other organs, soon appears. The older the patient, the severer is this secondary degeneration. This secondary degeneration of the arteries leads to ischaemic changes in the kidneys, heart, brain, retina and even limbs. Such arteries are also liable to rupture or thrombosis. Heart failure is a common result of cardiovascular hypertrophy in simple hypertension.

SYMPTOMS AND SIGNS

It is common experience to discover hypertension during routine clinical examination of a subject who is in good health and without symptoms. This state of well-being may persist for many years before symptoms peculiar to hypertension begin to appear, and when they do appear they may be so slight as not to cause appreciable discomfort. Often, however, the symptoms may be so severe as to produce distress far in excess of the signs elicited on physical examination. Thus, headache of a throbbing or bursting character may be

felt in the frontal or occipital regions. Giddiness, tinnitus, sleeplessness, nervousness, palpitation and left inframammary and scapular pain with tenderness are other common symptoms; such symptoms, singularly or severally, are common in healthy but nervous subjects and should not be regarded as specific symptoms of hypertension. Some degree of breathlessness is usual, but this can often be attributed to general weakness associated with the illness, or to obesity and general unfitness.

It is often possible to suspect hypertension from the increased force of the pulse, but it cannot be told with certainty and it is necessary to take manometric readings of the blood pressure. Increased arterial pulsation in the neck is



FIG 208—Hypertension which has produced much enlargement of the left ventricle (1), but only slight elongation of the aorta Hilar congestion (2) from failure

usually present, and sometimes it is noticeable, giving rise to the " kinked carotid " as a result of the raising of the aortic arch from elongation of the aorta The apex beat is displaced outwards in accordance with the degree of left ventricular hypertrophy which prevails. On auscultation there may be accentuation of the first and second sounds in the mitral area and of the second sound in the aortic area The first sound commonly shows splitting and, when this is conjoined with a loud first heart sound or a rough systolic murmur, it may resemble the presystolic murmur of mitral stenosis.

In some cases of simple hypertension, the *electrocardiogram* is nearly normal, but left electrical deviation is common. In the presence of much cardiac enlargement the R–T segment is depressed and the T wave in leads I and CR_7 is inverted as an index of left ventricular preponderance, or the curve is characteristic of left bundle branch block. When a patient with simple hypertension becomes subject to pain of cardiac ischaemia, the exclusion of cardiac infarction may sometimes be difficult if the T in lead I is inverted. When the T is inverted in IVR it is no proof of infarction If the inversion of the T wave in the chest lead CR_7 is greater than in IVR, it is likely that hypertension has not been complicated by cardiac infarction.

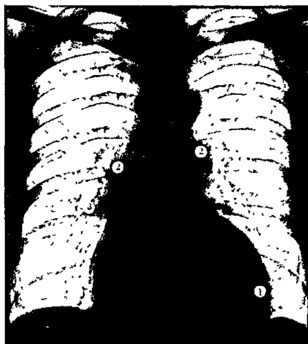


Fig 209—Hypertension which has produced much enlargement of the left ventricle (1) and unfolding of the aorta (2) Hilar congestion (3) from failure

The findings at *cardioscopy* are in two main groups according to whether the stress has fallen more heavily on the heart or on the vascular system. In the former there is much enlargement of the left ventricle, but in the latter, moderate or less enlargement of the left ventricle is associated with much elongation of the aorta, exaggerating the arc ordinarily produced by the aortic arch ; this effect is seen in both oblique positions, and in the left oblique position the *aortic window* is extended from displacement of the ascending aorta to the right and from elevation of the transverse arch which in turn reduces the height of the *aortic triangle* and lengthens its base (Figs 208, 209, 210 and 211)

These " cardiac " and " vascular " types of simple hypertension may be differentiated clinically as well as cardiographically, and the features which

characterize each are best summarized in Table VIII, but frequently both effects are seen in the same patient, and such might be named the "cardiovascular type."

TABLE VIII

Comparing the findings in the cardiac and vascular types of simple hypertension.

Feature	*Cardiac type*	*Vascular type*
Common specific symptoms	Heart failure	Vascular accidents
Auscultation	Systolic murmur common; fourth heart sound in failure	Normal, or splitting of first heart sound
Electrocardiogram	Left ventricular preponderance, left bundle branch block	Left axis deviation; some degree of left ventricular preponderance
Cardioscopy	Great enlargement of left ventricle	Prominent aortic elongation, moderate or slight cardiac enlargement

The other symptoms and signs of simple hypertension are best considered in relation to the anatomical distribution of the vascular effects (rupture, sclerosis, tortuosity, thrombosis, and spasm).

Epistaxis is a common and familiar symptom. Haemoptysis may be accepted as due to hypertension when the lungs have been found healthy on radiological examination and when the sputum contains no tubercle bacilli. Similarly, haematemesis should not be regarded as caused by hypertension until radiological examination after a barium meal has proved the absence of gastro-duodenal ulceration.

The cerebral artery may be the seat of spasm (hypertensive encephalopathy), or rupture (cerebral haemorrhage), or embolism resulting in cerebral infarction as the result of intracardiac thrombosis situated within an enlarged left ventricle.

Rupture of retinal arteries may involve the smaller tributaries, or may be extensive enough to produce detachment of the retina. Various degrees of tortuosity of the arteries are met with, compressing the veins at the points where they cross; the arteries also are narrow and may be pale and show an irregular outline. Such findings stress the importance of retinoscopy as a test in all patients suspected of having hypertension

Sclerosis of the smaller renal arteries may cause albuminuria, but not renal failure, although occasionally simple hypertension changes into papilloedemic hypertension as the result of necrotizing arteritis.



FIG 210—Hypertension producing great elongation of the aorta (1) in the left oblique position. Aortic window (2) enlarged Widening of the base of the aortic triangle (3) Only slight enlargement of the left ventricle (4) Left pulmonary artery (5) Male aged 60 years with a blood pressure of 230/120



FIG 211—Elongation of the aorta in hypertension (blood pressure 280/110) Right oblique view. Unfolding of aortic arch (1). Enlarged aortic window (2). Enlargement of left ventricle (3)

The limb arteries in simple hypertension do not usually produce symptoms The finding of thickened and whipcord-like radial and brachial arteries should be interpreted cautiously because it may be present in health. Even when it is identified with hypertension, and this finding should always be present in hypertension, the state of the peripheral arteries is never a sound guide to the state of the more vital arteries such as the coronary and the cerebral. Intermittent claudication from atherosclerosis of the femoral artery is probably no commoner in cases of hypertension.

Heart failure sets in stealthily in simple hypertension, unless ushered in suddenly by cardiac infarction, so that for some time the familiar signs of failure, cyanosis, distended veins of neck, crepitations at lung bases, distended liver, ascites and oedema of ankles are absent, and they only make their appearance when failure has been present for some time. Heart failure in a patient with hypertension has sparse signs and its presence is known when nocturnal breathlessness (cardiac asthma) appears ; triple rhythm from the addition of the fourth heart sound is then present on auscultation and particularly at the xiphisternum. Confirmation of the diagnosis is gained from the prominent diuresis induced by a reputable mercurial diuretic, and from congestion of the hilar regions seen on cardioscopy.

If the patient is seen at a later stage, when failure has advanced, the blood pressure reading may be normal or even low In this instance a diagnosis of hypertensive (biventricular) failure is made from the presence of some or all of the familiar signs of right heart failure, considerable enlargement of both the left heart and the right heart in the absence of valvular disease, and triple rhythm from the addition of the third heart sound, which is heard best over the displaced apex beat. In such a clinical picture the presence of cardiac infarction should be considered. Auricular fibrillation is sometimes found in hypertensive heart failure, and its response to treatment by digitalis may not always be as satisfactory as in the case of fibrillation in mitral stenosis.

TREATMENT

An appreciation of the natural history of simple hypertension in a large number of patients is necessary for the efficient management of individual cases. To realize the good prognosis over many years identified with the majority, is to guard against any excessive zeal in treatment, and it also serves to foster a judgement which condones a detached watchfulness over the patient, and represses any impulsive desire to try unproved remedies In no other condition has the therapist to be more mindful lest he yields to whims and insistent requests of his patient that "something should be done." If a raised blood pressure has been found in the course of routine clinical examination it is best not to inform the patient. When it accounts for symptoms the information may be shared, but from the start it should be accompanied by firm reassurance and judicial treatment of particular symptoms It is never permissible to disclose the actual blood pressure figure for it is likely that the patient will expect to know successive readings and in this way become morbidly preoccupied with the rise and fall of the blood pressure which is natural for the condition. It is obvious that a patient should not regard his symptoms as closely related to the height of his blood pressure When reassurance has helped to allay the patient's fear and to found a calm equanimity in its stead,

it is necessary to eradicate harmful influences if they are present. Thus, there might be a need to reduce physical or mental stresses associated with occupation, to forego excessive indulgence, and especially to adhere to a fat-reducing diet if obesity is present.

The drug treatment of simple hypertension has proved unsatisfactory. Of the

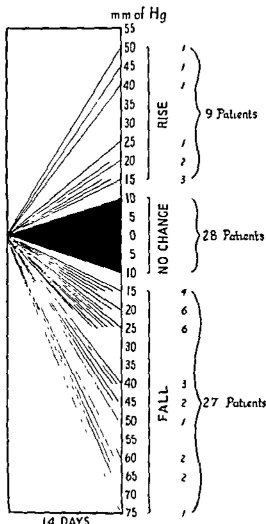


FIG. 212—Showing the change in the systolic blood pressure from the first to the second examination in 64 patients with hypertension. During an interval of a fortnight between the two records the patients were given a placebo mixture. A rise or fall of 10 mm. of Hg was not regarded as an appreciable change.

many drugs recommended there is not any agreement on which is the best to employ, and none has gained any outstanding reputation. When the effects on the blood pressure and symptoms, of thirty-three preparations were watched, and compared with those from a placebo in a series of patients, after allowance had been made for the tendency to obtain high blood pressure readings at the first

and possibly the second, examination (Fig. 212), and for the natural variation in the blood pressure values, none of the drugs produced hypotensive effects (Figs 213 and 214). The sedative drugs have value in temporarily relieving nervous symptoms when these are prominent, and half a grain of phenobarbitone given twice or three times daily according to need often proves helpful. These negative findings

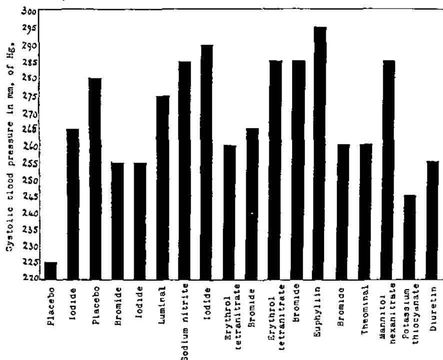


FIG 213—Natural variation in the blood pressure in a patient with hypertension during the administration for fortnightly periods of various preparations

with drugs in uncomplicated hypertension do not make it less desirable that the patient should remain under medical supervision, for timely and active measures have to be adopted when complicating cardiovascular effects of hypertension appear, notably heart failure

Surgical treatment by sympathectomy is under trial. It has sometimes produced great improvement, but often the results have been disappointing, and much time must elapse before the procedure can be evaluated. In this estimate of the benefit brought by this major operation, it is necessary to control the results and to take full cognizance of the longevity in patients under placebo therapy

PAPILLOEDEMIC HYPERTENSION

(*Synonyms*—Malignant hypertension or malignant form of essential hypertension, nephritis repens (type 4), chronic interstitial nephritis)

TERMINOLOGY

Papilloedemic hypertension may not be ideal terminology, but at least it removes

all uneasiness on the part of the medical practitioner and the patient during a discussion of the condition at the bedside. It also has the advantage of emphasizing its most characteristic physical sign

PATHOLOGY

A necrotizing arteritis involving the smaller arteries and arterioles of the kidney, adrenal body, pancreas, intestine, and less commonly of other organs, is the characteristic change at necropsy in this condition, along with cardiovascular hypertrophy and the effects of heart failure, or sometimes arterial rupture. The arterial necrosis is greatest in the kidney which is usually somewhat contracted,

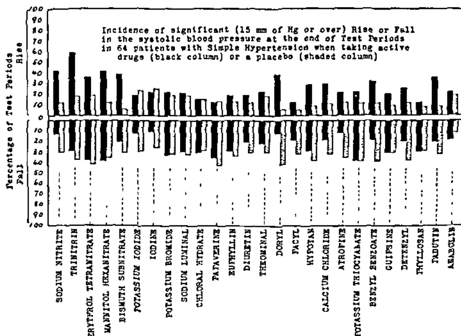


FIG 214—Comparing the variations in systolic blood pressure in 64 patients with hypertension which took place at the end of 14 days on placebo or active drug therapy.

and is specially common in the afferent arterioles to the glomeruli where the focal injury often produces obstruction of the arterial lumen. The interstitial tissue shows a varying degree of cellular infiltration and fibrosis. It is probable that arterial necrosis is the result and not the cause of the hypertension, for there is not any evidence that it is present during the earlier phase of the illness. The arterial lesions in the kidney eventually cause renal failure, but clinical signs of kidney damage are missing at the start, and only appear towards the end. On the other hand, it is likely that the renal vascular occlusions reinforce the hypertensive effects already present.

SYMPTOMS AND SIGNS

The patient, more commonly a male, is usually 40–50 years of age The symptoms, which are ushered in abruptly, are of three kinds. Morning headache may

be distressing and sometimes associated with nausea and vomiting. Increasing breathlessness, particularly nocturnal (cardiac asthma), signals the onset of left ventricular failure. Rapid failure of vision is the other characteristic symptom

All the physical signs common to simple hypertension are found in papilloedemic hypertension, but other special signs are added The systolic blood pressure does not differ materially, but the diastolic pressure is seldom less than 140, and it is commonly 160 or over. Pulsus alternans is commoner than in simple hypertension, but auricular fibrillation is less common. Cardiovascular hypertrophy is obvious and, as a rule, enlargement of the left ventricle is considerable. The addition of the fourth heart sound, initiating triple rhythm, is usual and is best heard near the xiphisternum ; this sign of left ventricular failure is the first to appear and precedes crepitations at the lung bases. Indeed, apart from this triple rhythm, evidence of failure is scanty, although the presence of pulmonary congestion is easily confirmed by the finding of dense hilar shadows at *cardioscopy*, which also shows considerable left ventricular enlargement. The *electrocardiogram* will show signs of left ventricular preponderance.

Retinoscopy is an invaluable test, because papilloedema in a case of non-renal hypertension signifies this more ominous variety. The sign is added to other retinal changes such as areas of haemorrhage and exudation, which are also met with in simple hypertension.

In the early stages of papilloedemic hypertension, examination of the *urine* may show only a small quantity of albumin, and, more rarely, none at all. As the disease progresses the albumin content increases somewhat, the specific gravity is lowered and becomes fixed, for there is polyuria, and the pigment decreases. The blood urea is raised and renal failure sets in, although this is often less noticeable than heart failure.

PROGNOSIS AND TREATMENT

The outlook in a case of papilloedemic hypertension is a grave one Cerebral haemorrhage is a likely complication from the start, and heart failure is a serious event which may appear early in the illness. With such a gloomy prospect the treatment is naturally palliative in kind and directed to symptoms as they arise. Hypertonic saline, rectally and intravenously, will often ease a distressing headache. In some cases, occasional venesection might be worth while. Repeated lumbar punctures have doubtful value in preventing or improving the retinal injury which is causing dimness of vision. Mercurial diuretics will alleviate heart failure symptoms for a time, and this therapy, intramuscularly, should be started whenever nocturnal dyspnoea is present, even although some degree of renal failure is appearing along with it. Lumbar sympathectomy is still under trial, but this much can be said for it, that in the meantime it is the one form of treatment which produces the greatest benefit for the longest period, and in the largest number of patients, but the measure is not curative The operation of choice is a bilateral supradiaphragmatic splanchnicectomy and lower dorsal sympathetic ganglionectomy.

RENAL HYPERTENSION

Experimental—Goldblatt showed that partial constriction of one or both renal arteries in the dog induced hypertension. The hypertensive effects of unilateral constriction were more severe when the opposite healthy kidney had been removed.

By the same method Wilson and Byrom produced hypertension in rats and studied its effects on the unclamped healthy kidney. They found changes similar to those taking place in papilloedemic hypertension in man. In these experiments the severity of the hypertension varied with the degree of arterial constriction. Moderate constriction produced a state comparable with simple hypertension in many, whereas greater constriction induced vascular changes similar to those found in papilloedemic hypertension, and such changes were found in all organs except in the clamped kidney which had been protected from the effects of hypertension.

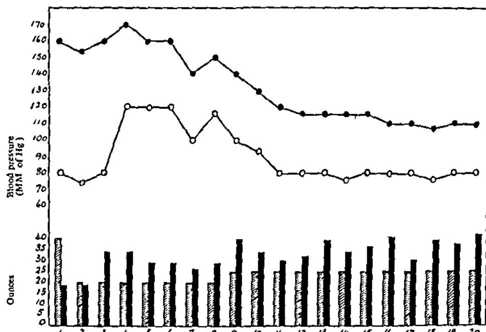


FIG 215 —Temporary hypertension and heart failure in acute nephritis. Return of the blood pressure to normal, and diuretic response, following rest in bed on a diminished fluid intake Systolic blood pressure is indicated by dark line and the diastolic blood pressure by lighter line. Black columns indicate urinary output and shaded columns fluid intake in ounces, plotted daily

Removal of the ischaemic kidney annulled the hypertension so long as vascular changes had not taken place in the other kidney, for they in themselves were found capable of maintaining the ischaemia and the hypertension. Ischaemic hypertension has been modified and often removed by ligation of the ureter Although the work in experimental renal ischaemic hypertension has been of a high order, the actual mechanism is not yet completely understood. It is likely that a pressor substance (renin) is liberated by the renal cortex and combines with a plasma protein (hypertensinogen or angiotonin activator) to form hypertensin or angiotonin. Intravenous injection of hypertensin causes an immediate rise in blood pressure, its action is reduced in the presence of normal kidney tissue which yields an antipressor enzyme (hypertensinase or angiotonin inhibitor).

Hypertension in nephritis.—From a study of the natural history of patients with Bright's disease and histological examination of those coming to necropsy, Ellis

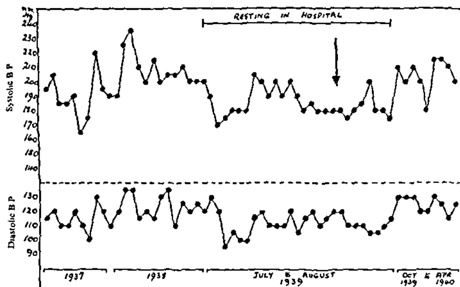


FIG 216.—Record of blood pressure in a female aged 57 years with simple hypertension Arrow indicates removal of left kidney. The kidney was structureless from great atrophy, and showed numerous cavities and fibrous obliteration of the ureter.



FIG. 217.—Records of blood pressure in a female aged 51 years with simple hypertension Arrow indicates removal of right kidney The kidney showed no gross macroscopical changes, but microscopy demonstrated ischaemic atrophy of tubules and glomeruli from arterial narrowing.

described two types of nephritis. In the *first type* the onset of the illness is abrupt, general symptoms are present and so are haematuria and oedema. A history of previous infection such as a sore throat is common and recovery is usual During the early part of the illness there is moderate or even greater hypertension, and hypertensive encephalopathy may occur, but as a rule on a restricted fluid intake the blood pressure returns to normal limits by the beginning of the third week (Fig 215) Hypertension is a feature of those cases which show symptoms of heart failure during the illness. Less commonly the hypertension may linger for years as the condition moves into its chronic form and, rarely, it may rise abruptly as in the terminal phase of the next group In the *second type* of nephritis the onset is more insidious, general symptoms are absent and so is haematuria usually; oedema persists A history of previous infection is uncommon and recovery seldom takes place The blood pressure at the start of the illness may be normal or only slightly raised, but eventually it rises sharply with the onset of renal failure. Indeed, the state of the blood pressure in type II nephritis is an index of prognosis. Thus, when the blood pressure is high at the start, the survival period is no longer than from one to five years. On the other hand, if the blood pressure is only slightly raised the illness is likely to be a protracted one with a rise of blood pressure taking place much later.

Hypertension in toxaemia of pregnancy —Most patients with toxaemia of pregnancy recover completely after delivery, but in some, hypertension and albuminuria persist. If hypertension is not severe the patient may pass successfully through further pregnancies Eventually the blood pressure rises, and renal failure and heart failure appear at the end. The hypertension associated with toxaemia of pregnancy appears to be of the simple kind, except that albuminuria is more frequent and severe, and renal involvement with renal failure more common.

Hypertension in unilateral renal disease.—Since Goldblatt's work on renal ischaemia it was natural for clinicians to seek examples of unilateral renal disease amongst their patients with hypertension and to watch the effects of nephrectomy. The unilateral lesions found in association with hypertension might be atrophic pyelonephritis, hydronephrosis, pyonephrosis, calculus, and vascular renal lesions. Although many satisfactory cases have been reported, in the greater number nephrectomy has not annulled the hypertension (Fig 216), it is not yet known in these unsuccessful cases whether vascular lesions had already taken place in the remaining kidney which maintained the state of hypertension after removal of the ischaemic diseased kidney as in the experimental animal.

Even in simple hypertension, radiological examination of the renal area after intravenous Uroselectan may show a slight delay on one side. When such a kidney has been removed the hypotensive effects have been temporary (Fig. 217), again suggesting that vascular changes are present in the remaining kidney, although probably less than in the extirpated one.

It is natural to assume that a greater measure of success is expected in those cases in which the unilateral renal disease is early and in which the resulting hypertension has not been established long enough to injure the opposite healthy kidney. Such examples, however, are uncommon and the search for them has been disappointing.

CHAPTER 11

THE HEART IN DISEASE OF THE LUNGS

IN PULMONARY EMBOLISM

A SMALL pulmonary infarction does not affect the heart, and does not show any abnormality in the cardiographic tests; it is the massive pulmonary embolism that causes obvious changes

SYMPTOMS

Venous thrombosis, commonly in the lower extremities, may be apparent before the episode, but usually no such warning is present, and severe dyspnoea, distress, and a feeling of suffocation in the chest, are ushered in suddenly, probably some days after an uneventful surgical operation. The pulse is rapid and small, and the blood pressure falls. On auscultation the presence of triple rhythm from the addition of the third heart sound is a valuable sign, telling of failure of the right heart The patient may not survive long enough for a cardiographic examination to take place, but should tests become available, they are characteristic of right heart failure from obstruction within the pulmonary circulation (cor pulmonale)

At the commencement the *electrocardiogram* usually shows right bundle branch block, and soon this gives way to inversion of the T wave in leads II and III, and CR_1 (Fig. 218) The Q wave is often prominent in lead III, as is the P wave in leads II and III. The tracing is told from that typifying posterior cardiac infarction by the deep S in lead I, inversion of the T in CR_1, and an upright T wave in CR_7

Cardioscopy in the early phase of the illness will show enlargement of the right heart and later, during recovery, a lessening of the cardiac shadow may be demonstrated (Fig. 219). The lung fields may show an opacity at the infarcted area but, rarely, the lung may escape infarction.

IN EMPHYSEMA

As a rule, the breathlessness associated with emphysema signifies a reduction in pulmonary ventilation through a fault of the true respiratory mechanism, and is not an expression of heart failure Indeed, heart failure may not be a feature of emphysema for many years, and when it does set in it is a serious event. No changes in the pulse are identified with emphysema, although the rate is often more rapid. Hypertension may be present, and this finding influences prognosis adversely, because it contributes to failure

Size of the heart in emphysema—Noticeable enlargement of the heart from emphysema is uncommon, although there are exceptions The mechanism determining such enlargement has evaded explanation so far. In the meantime, cases of emphysema may be arranged with advantage in four groups according to the degree of cardiomegaly; such a division helps in assessing prognosis in individual patients.

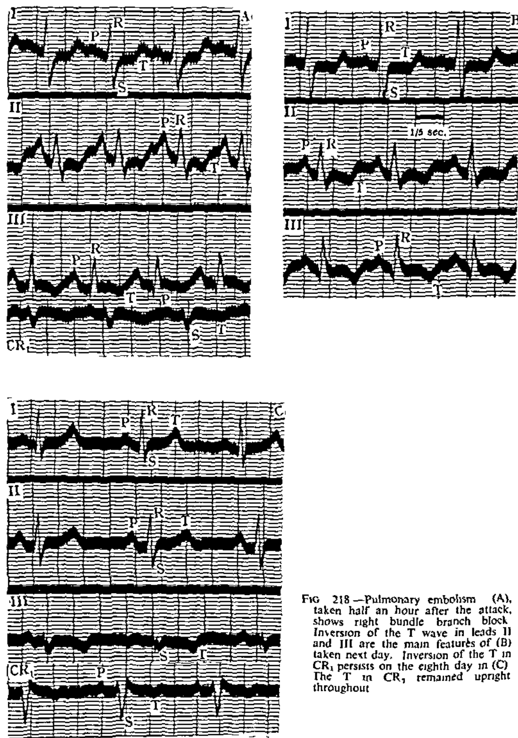


FIG 218—Pulmonary embolism (A), taken half an hour after the attack, shows right bundle branch block Inversion of the T wave in leads II and III are the main features of (B) taken next day. Inversion of the T in CR_1 persists on the eighth day in (C) The T in CR_7 remained upright throughout



FIG 219A



FIG 219B.

FIG 219—Enlargement of the heart from massive pulmonary embolism (B) and (C) (which appears overleaf) were taken at fortnightly intervals following (A), which was recorded a week after the embolism.

In the *first group* there is no visible cardiac enlargement. Here, breathlessness and productive cough from emphysema and bronchitis, might have been present, with seasonal variation, for many years The apex beat is not displaced outwards, and the first and second heart sounds are usually best heard in the mid-line at the xiphisternum ; triple rhythm is absent. Electrocardiographic abnormalities are absent or slight, and on cardioscopy there is no enlargement of the heart (Fig. 220). Such patients do not present clinical evidence of heart failure, and the terminal



FIG 219C.

illness, like the symptoms occasioning ill-health over many years, is of pulmonary rather than cardiac origin, and is often in the form of bronchopneumonia.

The *second group* also shows no visible enlargement of the heart. The symptomatology resembles that of the first group, but there are added two characteristic signs, namely, cyanosis and distension of the pulmonary artery on cardioscopy (Fig. 221).

In the *third group*, slight or moderate enlargement of the right heart may be seen on cardioscopy, and some cardiographic evidence of right heart preponderance may also be present. The clinical course of patients in this group may be the same as those in the first group, but some evidence of heart failure may accompany the terminal pulmonary complications, although it is seldom uppermost in the clinical picture.

In the *fourth group* there is obvious, but seldom considerable, enlargement of the right heart, and the signs of heart failure are present Breathlessness in such patients is associated with some degree of cyanosis, distension of veins in the

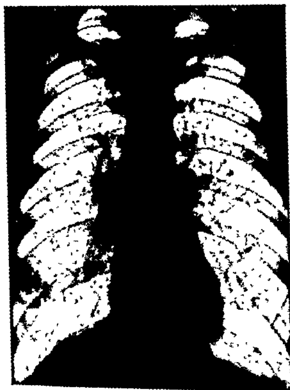


Fig. 220.—Emphysema. No enlargement of the heart nor of the pulmonary artery

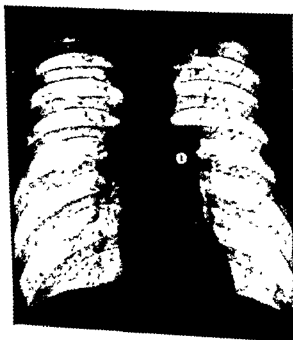


Fig. 221.—Emphysema. No enlargement of the heart, but the pulmonary artery (1) is very prominent

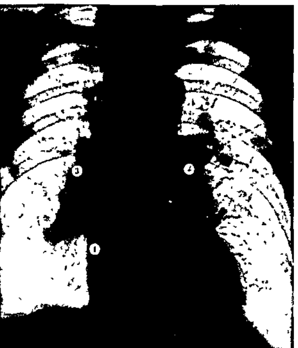


Fig 222.—Heart failure in emphysema. Enlargement of the right auricle (1), and of the pulmonary artery (2); great pulmonary congestion (3). Triple rhythm was present on auscultation from addition of the third heart sound.

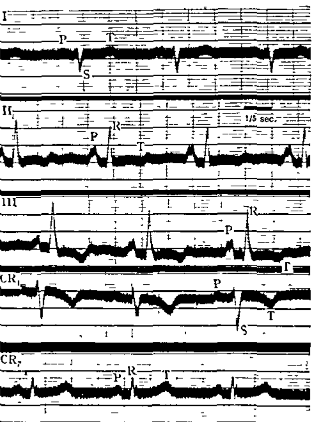


Fig 223.—Heart failure in emphysema. Right axis deviation and right heart preponderance instanced by inversion of the T wave in leads II, III, and CR_1; the T wave is upright in CR_7.

neck, crepitations or hydrothorax in addition to the signs of bronchitis and emphysema, enlargement of the liver, ascites, and oedema of the ankles. A triple rhythm from addition of the third heart sound is usually found over the displaced apex beat. On cardioscopy, in addition to the signs of emphysema, there is present some enlargement of the right auricle, right ventricle, and pulmonary artery, together with much pulmonary congestion (Fig. 222) The electrocardiogram does not always show the distinctive changes from heavy right heart preponderance (Fig. 223) with a deep S wave in lead I and inverted T wave in leads II, III and CR_1, and the signs of right ventricular preponderance are less well

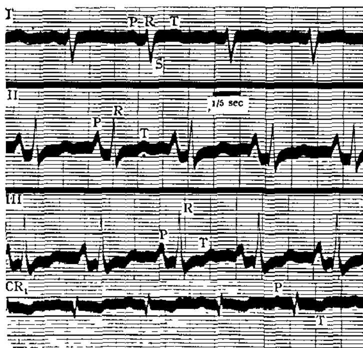


FIG 224 —Heart failure in emphysema Right axis deviation Early right heart preponderance instanced by a diphasic T wave in leads II and III, and inverted T in CR_1, the P wave is *tall in II and III.*

formed (Fig 224) Whenever the clinical and cardiographic signs, common to this group, have been collected in a patient with emphysema, survival cannot be for longer than a few months.

IN PULMONARY HYPERTENSION

Pulmonary hypertension has been described as primary or secondary. It is secondary when caused by certain cardiac and pulmonary disorders including diseases of the pulmonary arteries, like syphilitic arteritis, which is dependent on extrinsic factors. *It is primary when these conditions* are absent, and when vascular disease, if present, is not clearly dependent on any extrinsic factor. In a recent case of primary hypertension we found stenosis and occlusion of the

muscular pulmonary arteries by endarteritis fibrosa. The endarteritis resembled that found in the systemic arteries in systemic hypertension, and was also probably secondary to hypertension and, in this instance, of the pulmonary arterial system. Foci of medial aplasia or hypoplasia in the arteries numbered many thousands. It is likely that endarteritis formed over some of these deficiencies during attacks of mild hypertension, such as might have been produced by coughing ; subsequently the resulting stenosis or occlusion led to persistent hypertension The primary abnormality leading to the hypertension, therefore, was deficiency of development in the media of the pulmonary arteries

SYMPTOMS AND DIAGNOSIS

Breathlessness, cough and cyanosis are the outstanding symptoms of primary hypertension The signs of right heart failure are present including distension of

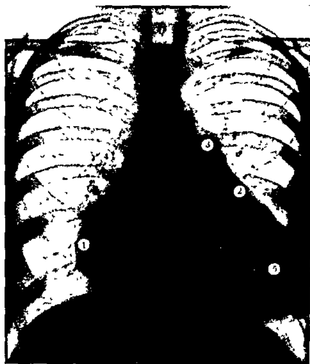


Fig. 225 —Primary pulmonary hypertension Great enlargement of right auricle (1), conus (2), and pulmonary artery (3) Much pulmonary congestion (4) Left ventricle (5) normal and displaced to left by enlarged right heart. Female aged 49 years (necropsy control)

cervical veins, sometimes crepitations at the lung bases, enlargement of the liver, ascites and oedema. Triple rhythm due to addition of the third heart sound may be present over the apex beat which is displaced outwards by enlargement of the right heart. On *cardioscopy* (Fig. 225) such enlargement is seen to involve the right auricle, right ventricle, and pulmonary artery ; pulmonary congestion is also present. The changes due to emphysema are absent. The *electrocardiogram* is characteristic of right heart preponderance (Fig. 226), showing a deep S wave in

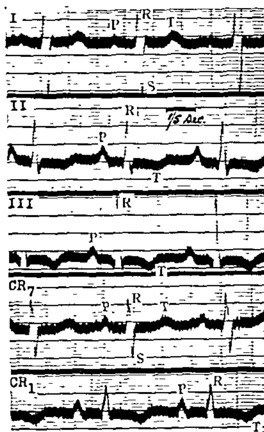
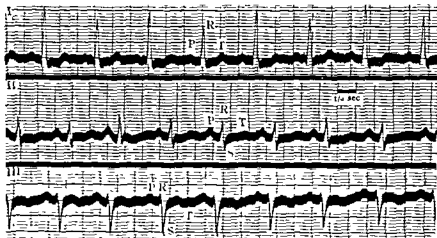


FIG 226.—Primary pulmonary hypertension Right axis deviation Right heart preponderance as instanced by inversion of the T waves in leads II, III and CR_1

FIG 227 —Electrical alternans in lead I in a patient without heart disease apart from a moderate hypertension



lead I, and inversion of the T wave in leads II, III, and CR_1. The cardiogram is told from that which characterizes posterior cardiac infarction by the prominence of S_1, inversion of the T in CR_1, and the upright T in CR_7.

IN PNEUMONECTOMY AND PNEUMOTHORAX

Arrhythmia, particularly auricular fibrillation or auricular tachycardia, is not uncommon after a surgical operation on the chest involving pneumonectomy or directed to the radical treatment of carcinoma of the oesophagus. Age appears to be a predisposing factor, for it is rare to meet with arrhythmia in such cases below the age of 40 years. It is thought that vagal irritation from mediastinal displacement or from infection, might be the cause of the abnormal rhythm. It is best to restore normal rhythm as soon as practicable by the administration of quinidine.

No serious cardiac effects result from pneumothorax, but innocent cardiographic irregularities such as electrical alternans (Fig. 227) are sometimes recorded.

CHAPTER 12

HEART FAILURE

THE MECHANISM of heart failure has been debated through the years Controversy has mainly concerned two theories. The *back-pressure* theory envisages stasis of the blood in the vascular areas which drain in the direction of the affected heart chamber. Thus, in mitral stenosis the sequence of events may be distension of the left auricle, the pulmonary vessels, the right ventricle and auricle, engorgement of the veins of the neck with cyanosis, and of the portal and lower systemic venous systems, producing enlargement of the liver, ascites, and oedema of the ankles. The *forward-pressure* theory postulates a diminished output of blood from the heart, resulting in stasis in the vascular areas placed in front of the affected heart chamber, and in ischaemia of the tissues. It is likely that both back-pressure effects and a diminished cardiac output operate in many, if not in most, cases of heart failure. Fortunately, however, the *mechanism* of heart failure need not concern us unduly, for the progress of events in the different heart lesions during failure is known and the exact manner in which they arise is of lesser importance.

CLASSIFICATION

Patients with heart failure may be grouped according to the specific symptoms they present or the response they make to individual remedies. Thus, cases may be arranged in two classes according to whether heart failure affects chiefly the left or the right side of the heart, this will apply particularly when discussing hypertensive heart failure. Another classification suggests dividing the cases into two groups, according to whether the failure is associated with normal or abnormal rhythm. When applied to treatment this second classification implies that mercurial diuretics produce great benefit in the first group and that digitalis, although essential for the first group, excels in the second. No single classification, however, is satisfactory by itself, and it is necessary to discuss the peculiar symptomatology and treatment of heart failure found in the various heart conditions giving rise to it.

SYMPTOMS AND SIGNS

*The familiar symptoms of established heart failure are breathlessness, cyanosis, distension of cervical veins, crepitations at the lung bases or the signs of hydro-*thorax, distension of the liver, ascites, oedema of the ankles, and scanty urine with albuminuria. To wait for this comprehensive clinical picture to develop, however, is to delay effective treatment, particularly if the failure affects primarily the left ventricle The need for recognizing early heart failure is only equalled by the need for early treatment which inevitably depends on early diagnosis In order to gain this end the earliest evidence of failure should be sought in those patients known to suffer from heart disease which is expected to give rise to heart failure sooner or later. In such patients symptoms develop gradually and our awareness of this event should lead us to seek the earliest signs of failure, and we should not *wait for the fully developed picture.*

Breathlessness is the first signal of heart failure. Shortness of breath on exertion occurs in health ; with advancing age, especially in the presence of obesity, this natural reaction to increased physical performance is expected. The breathlessness which heralds heart failure is characterized by its occurrence during exercise which hitherto produced no breathlessness, or by dyspnoea in excess of that customarily induced by a standard form of exercise. Thus, pathological breathlessness is present when it is induced by exercise which usually produces none, when exercise which previously initiated breathlessness causes it in undue measure, and when breathlessness has increased during a short period of time. Nocturnal breathlessness (cardiac asthma) is characteristic of left ventricular failure.

An increase in the venous blood pressure may be regarded technically as the first evidence of heart failure, but its estimation is too difficult to become common practice in clinical medicine. The extent of *venous engorgement* in the neck has not always proved satisfactory as a clinical measure of the degree to which the venous pressure has risen as the result of right heart failure, but it is worthy of notice. In health, all veins which lie at a higher level than the manubrium sterni remain collapsed and those below are distended. Thus, when a healthy subject is in the reclining posture with the head resting on a pillow, the external jugular veins are distended for about one-third of their course from the clavicle to the angle of the lower jaw. In a patient with a slightly raised venous pressure the column of venous distension reaches the centre of the sternomastoid muscle or higher This clinical indicator of the venous pressure should gain significance not on account of its accuracy, but on account of the simplicity of the test.

When persistent crepitations are elicited over the lung bases in heart failure, considerable *pulmonary congestion* can be presumed present. Early evidence of pulmonary engorgement is marked by vascular congestion in the hila of the lungs, but this evades clinical auscultation and is only discovered by cardioscopy. The picture obtained is a characteristic one, presenting wing-like shadows spreading from each hilum, and caused by an increase in the density of the pulmonary vessels through engorgement , as the process increases the vessels may be seen here and there in cross-section and appear like ink blots. Occasionally the lung fields are more diffusely involved from pulmonary oedema. The help provided by cardioscopy in the recognition of early heart failure has not yet gained the reputation it deserves. Nor should the method be applied solely in diagnosis, for it can prove useful in watching the progress during treatment, since the extent of hilar congestion determined at intervals by cardioscopy can tell the advance or retreat of the process of failure (Fig. 228).

Hepatic distension is a sensitive indicator of an increase in the lower systemic venous pressure, so that whenever right heart failure is suspected, enlargement of the liver should be sought Palpation immediately below the right costal margin may elicit tenderness in a patient with hepatic distension, but a quest for the descending liver edge as the hand travels upwards from below in the right iliac fossa is a more reliable method of examination ; in the presence of ascites the " dipping " method of palpation is more likely to reveal the size of the underlying liver.

The *size* and *position* of the heart in failure need to be examined. Cardiac enlargement is usual in failure, but it is seldom the direct result of failure and its

distribution and extent depend chiefly on the lesion which is providing the cause of the failure. Either with or without failure the progression of cardiac enlargement is very gradual, so that sudden dilatation of the heart seldom occurs. Whenever such an event has been implied from a clinical examination and displacement of the apex beat to the left has been noticed, influences contriving

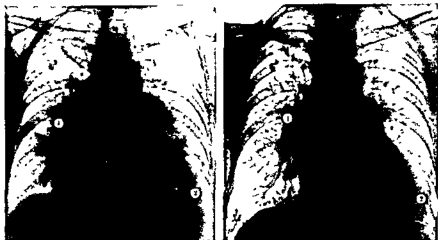


FIG 228A & B



FIG 228C

FIG 228 — Hypertensive heart failure Recession of pulmonary congestion (1) during treatment with rest and mercurial diuretics (A) was taken before treatment commenced, (B) one week later, and (C) a month later Enlargement of the left ventricle (2)

to produce this change must be kept in mind before attributing it to cardiac enlargement They include: tachycardia, with a thrusting apex beat, distension of the liver and ascites causing elevation of the diaphragm, and right-sided hydrothorax. Even on cardioscopy caution is necessary when assessing the development of cardiac enlargement, so that the squatting posture of the heart, assumed as a result of a raised diaphragm from any cause, must not be interpreted as enlargement. Again, the possibility of pericardial effusion enlarging the cardiac silhouette

should receive attention before an increase in the size of the heart is diagnosed in a case in which an exacerbation of failure has taken place with the formation of effusion within serous cavities.

The special symptomatology identified with certain heart conditions during failure will be described later when these are dealt with separately, but it may be mentioned here that in heart failure from pulmonary stenosis, emphysema, cardiac infarction, or thyroid toxaemia, enlargement of the heart is seldom conspicuous and, as a rule, it is only moderate.

Oedema of the ankles is a common finding in failure of the right heart, but it needs emphasis that it is absent in left ventricular failure, and in this condition only sets in when failure has persisted for some time. Oedema in the presence of breathlessness from obesity or bronchitis is too often attributed to heart failure.

Although *jaundice* in heart failure has sometimes been attributed to the absorption of the products of pulmonary infarcts, its cause is usually found in the distended and congested liver *Albuminuria* is a common finding in heart failure.

TREATMENT

Pre-failure stage

A patient with heart disease is commonly examined during the pre-failure period, and at that time he or she will derive greater benefit from observation and instruction than from the initiation of any form of zealous treatment. Medicinal therapy, apart from a placebo on occasion, is unlikely to produce material improvement during this period Certainly any attempt at digitalisation at this stage is ill-advised and without promise of benefit, so that it should be discouraged even though it might be given without great harm. In particular the patient should be instructed about physical activities and diet. Although it is permissible and even desirable to indulge in limited exercise, exertion should be forbidden. Activities connected with the patient's occupation and domestic duties need to be assessed before reviewing the possibility of sharing in games as well. In connexion with diet, this should not be heavily salted, never large or indigestible and, if obesity is present, meals should be small and of the fat-reducing kind. Daily fluid intake should not exceed $2\frac{1}{2}$ pints. To gain the co-operation of the patient during this pre-failure stage should be a first consideration. No amount of enthusiasm in treatment will produce improvement if the patient is not the doctor's ally. Nor must the psychological side be neglected, so that encouragement and just optimism should be dispensed along with firm dissuasion when needed. The over-cautious should receive encouragement and the reckless should be restrained and be made to realize the consequence of any disregard of instructions Improved health should be promised on condition and as a reward for attention to advice. If the signs of early heart failure are beginning to collect, and there is increased breathlessness with hilar congestion detected by cardioscopy, apart from the precautions already enumerated regarding exercise and diet, periods of controlled rest should be outlined ; these might be in the morning, after the mid-day meal, and at the week-end. The treatment adopted for this group of patients almost merits the designation of prophylactic treatment ; at any rate, it aims at postponing the signs of established heart failure. The

measure of success gained from the care devoted to these patients justifies the zeal involved in collecting cases within the scope of this group.

Abrupt heart failure

The precipitation of severe heart failure symptoms is sometimes unexpected, and this is met with particularly when cardiac infarction complicates hypertension. Symptoms in cases of established heart failure, hitherto controlled by treatment, may also progress suddenly during a period of increased physical activity or from the onset of some intercurrent illness. It is in this class that the urge to do something is so strong, and good judgement so frequently yields to mismanagement created by over-enthusiasm. The term *cardiac stimulant* has been applied to a drug the purpose of which is to help the heart in this crisis Many medicines have gained a reputation in this field, but usually it has been unmerited and founded on casual and uncontrolled clinical impression. Strychnine, camphor, coramine, and pituitary, are in this category and have far survived their usefulness in this field. A warning is overdue concerning the danger of wholesale and indiscriminate use of proprietary preparations imputed to be of this class of cardiac stimulants by virtue of their action variously described as taking place in the medulla, peripheral circulation, coronary circulation, or acting mysteriously as cardiac hormones. A plea for resort to controlled clinical assay is made in order to gain proper perspective of the effectiveness of these potions. Strophanthin, too, need not be used for it has seldom shown superiority over digitalis Whenever abrupt heart failure is associated with auricular fibrillation of high ventricular rate, rapid digitalisation is advisable and it is best carried out by giving 2 mg. of digoxin by mouth If cyanosis is prominent and accompanied by conspicuous venous engorgement in the neck, venesection is good procedure If restlessness and sleeplessness are present, hypnotics should be given in adequate doses after providing suitable support for the shoulders. In order to gain this comfortable posture for the patient and to facilitate nursing, it is desirable to acquire a specially devised bed Inseparable from any method adopted to ensure rest in a case of heart failure is the alleviation of the patient's natural apprehension. Alcohol, in that it is conducive to sleep and causes a feeling of well-being, may be dispensed with benefit, but in no sense should it be regarded as a heart tonic or stimulant Phenobarbitone may also contribute to the same end. Breathlessness is naturally present and it is usually severe, so that morphine (¼ grain) should be given on two or three successive nights, but it needs emphasis that a mercurial diuretic should be given at the earliest opportunity, for only when efficient diuresis has been induced on some two occasions will the relief of pulmonary congestion remove the breathlessness and permit sleep In the case of a female patient with abrupt heart failure it may be necessary to anticipate the considerable diuresis from a mercurial salt by tying a urinary catheter in position so as to avoid the exertion incidental to a frequent use of the bed-pan. When the urgent symptoms connected with abrupt heart failure have abated, further treatment with digitalis and mercurial diuretics may be planned according to the underlying lesion causing the failure and the nature of the heart rhythm.

Established heart failure

This designation is applicable when characteristic physical signs of failure develop gradually and progress during the absence of treatment or with inadequate

treatment. No rigid rules can be laid down to govern the management of all cases of established heart failure, and every patient must be studied individually, but in general the treatment should provide for sufficient rest, efficient diuresis, and adequate digitalisation.

Rest.—The object of rest in the treatment of heart disease is to ensure that physical activity does not add to the burden of a failing heart, so that the resting period need not often be too prolonged or too rigidly applied. When oedema has disappeared and breathlessness is no longer incurred during the process of dressing and undressing, ambulatory treatment is to be preferred to complete rest. In many patients the necessity to follow a period of resting in hospital when heart failure sets in comes from the need to educate them and instruct them on the

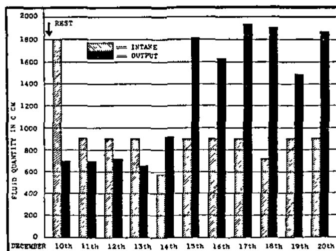


FIG. 229.—Chart showing diuresis following rest in bed and a diminished fluid intake in patient with mitral stenosis and heart failure with normal rhythm.

contingencies (rest, diet, and fluid intake) which are so essential to augment the drug treatment of heart failure. It is good practice for patients in hospital, undergoing treatment for failure, to be observed for at least a fortnight during ambulatory or convalescent treatment before discharge to their homes. Nor should the observation of progress stop here, for an almoner (already known to the patient in hospital) should visit the home and obtain knowledge about the patient's financial status, ability to take adequate rest, and to ensure that other members of the family have a practical and sympathetic understanding of the illness.

Diuresis.—To produce efficient diuresis should be a constant aim in every case of heart failure and there is more than one method available to attain it. That rest in bed combined with a diminished fluid intake can produce prominent diuresis has not gained sufficient recognition. Figure 229 illustrates this effect in a female patient aged 36 years, with mitral stenosis, in whom symptoms of early heart failure presented. Cognizance of this happening is necessary for the evaluation of any remedy credited with diuretic properties; in the patient reported

here, unmerited repute might have been accorded any drug professing a diuretic action if it had been introduced at the time the patient was admitted to hospital,

TABLE IX

Showing the development of uraemia in a patient with hypertensive heart failure during effective diuresis from a mercurial salt, diminished fluid intake and heavy doses of ammonium chloride.

Date	*Urinary output in ounces after 1 cc. of Salyrgan, when 30 grains of ammonium chloride was given three times daily*	
	Intravenously	*Intramuscularly*
December		
1	121	
4		158
7	133	
10		152
17	165	
20		172
23	140	
27		133
31	129	
January		
3		102
7	104	
10		102

14	Efficient diuresis followed Salyrgan suppository.
19	Patient became very ill, with weakness, listlessness, anorexia, drowsiness, sickness, and dry mouth and tongue.
21	Ammonium chloride and Salyrgan discontinued and fluid intake increased.
22	Blood urea 0 178 per cent.
25	" " 0 225 " "
27	" " 0 115 " "
31	" " 0 109 " "
February	
7	" " 0 090 " "
13	Patient left hospital feeling well and free from symptoms.

when she was voiding scanty urine The lesson in this connexion is two-fold; first, it emphasizes the need to restrict fluid intake in patients with heart

failure to $1\frac{1}{2}$ pints (30 ounces) daily at the start; secondly, it illustrates the importance of adequately controlling all clinical tests conducted to assess the diuretic properties of any drug. When a patient with heart failure has made progress and has become ambulatory, the intake of fluid may be increased to 2 pints (40 ounces) daily, and even more in the case of elderly patients, for in these the threat of uraemic symptoms, uncommon though it is, during the continuous use of diuretics and a diminished fluid intake, should be kept in mind (Table IX). Since vitamin C possesses diuretic properties (Fig 230), it is

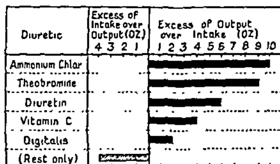


FIG 230—Average daily excess of urinary output over fluid intake in 9 patients with anasarca when treated with vitamin C and 4 reputable diuretics during a period of three weeks This is compared with the adverse fluid balance present when medicine was not given

necessary to include in the diminished fluid intake an adequate amount of the vitamin and provision for this is made in the fluid diet outlined in Table X. In order to gain the same end, the solid diet should not be heavily salted. A strictly salt-free diet allowing an increased fluid intake is not likely to displace in practical therapeutics a moderate salt-free diet and a diminished fluid intake because it cannot be tolerated for long by the patient.

TABLE X

Proposed daily fluid diet of 30 ounces for a patient with heart failure.

Meal-time	*Nature of fluid*	*Quantity (in ounces)*
7–8 a.m (breakfast)	Tea	5
10 a.m.	Tea or milk	6
12 30 p.m (dinner)	Lemon juice	5
3.30 p.m. (tea)	Tea	6
6.30 p.m. (supper)	Milk	4
8 p.m.	Orange juice	4

Mercurial diuretics.—Digitalis is without equal in the treatment of heart failure in mitral stenosis with auricular fibrillation, though diuretics may also be needed. When heart failure occurs with normal rhythm, as in hypertension, mercurial diuretics are indispensable and should be given with digitalis to increase the urinary output, lessen pulmonary congestion, and relieve dyspnoea. The

circumstances that demand the use of diuretics are known, but it is necessary to decide which preparation is best to prescribe, in what form to give it, and how best to improve upon its own diuretic effect. When the diuretic effects of five different preparations were compared in a series of patients, Neptal (May and Baker) proved to be one of the best. When the results of intravenous injection were tested side by side with intramuscular injection, with few exceptions the intravenous method produced greater diuresis (Table XI) and it possessed

TABLE XI

Comparing the diuretic effects of intravenous and intramuscular injections of four mercurial salts. Numerals denote the diuretic index, which expresses urinary output in ounces and in excess of fluid intake.

Case No.	*Preparation*	*Intravenous injection*		*Intramuscular injection*	
		Same day	*Next day*	*Same day*	*Next day*
3	Esidrone	76	0	57	14
4	Salyrgan	73	20	40	12
6	Neptal	176	10	119	1
7	Salyrgan	57	3	48	−8
8	Mersalyl	37	10	24	52
10	Neptal	83	−1	69	2
10	Salyrgan	83	−4	42	−6
12	Neptal	26	32	58	39
12	Salyrgan	76	50	28	38
18	Salyrgan	32	3	62	−11
21	Mersalyl	65	10	56	10
22	Esidrone	116	10	87	21
22	Neptal	84	16	38	−13
23	Neptal	57	5	47	0
24	Esidrone	96	9	62	−13
Average diuretic index		76	11	56	9

the additional advantage of being less painful to the patient Of the two kinds of rectal suppositories which were tried, Novurit produced much the better results, but it was inferior to Neptal given by mouth in tablet form (Fig 231) Thirty grains of ammonium chloride given two hours before the administration of a mercurial salt proved to be the best form of premedication to increase the diuretic effect of the mercurial preparation (Table XII) Ammonium chloride should always be dispensed in a palatable form and as enteric sugar-coated tablets each containing 7½ grains In summary, it may be said that in a patient confined to bed with heart failure and especially with oedema, standard treatment should include the intravenous injection of a mercurial diuretic (2 cc) twice weekly at the start and later once a week, preferably preceded on each occasion by the administration of four enteric sugar-coated tablets (30 grains) of

ammonium chloride by mouth two hours before. Before leaving hospital a trial should be given to three tablets of Neptal by mouth after the same premedication with ammonium chloride, if this produces a moderate diuresis it will be convenient to use tablets once a week when the patient leaves hospital so that the number of injections can be reduced.

Since mercurial diuretics are so commonly employed in the treatment of heart failure, it is necessary to emphasize the rarity of untoward effects when these

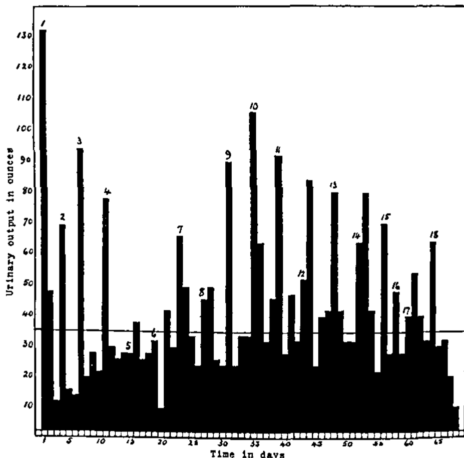


FIG 231 —Diuresis produced by different mercurial diuretics administered in different ways and 2 hours after ingestion of 30 grains of ammonium chloride to a patient with hypertensive heart failure during 10 weeks in hospital. Daily fluid intake was restricted to 35 ounces (1) Salyrgan intravenously (iv) (2) Mersalyl (iv) (3) Neptal (iv) (4) Esidrone (iv) (5) Novurit suppository (s). (6) Novurit (s) (7) Neptal by mouth (m) (8) Salyrgan (m) (9) Neptal (m) (10) Neptal (m) (11) Neptal (iv) (12) Neptal (m) (13) Neptal (m) (14) Neptal (m). (15) Salyrgan (m). (16) Mersalyl (m) (17) Mersalyl (m) (18) Neptal (m)

are considered alongside the fact that this therapeutic measure is used with success and safety in such a large number of patients Two precautions need to be kept in mind, however, in the treatment with mercurial diuretics of patients presenting oedema. The *first* concerns cases of heart failure in which the diuretic response, previously good, begins to fail and oedema increases; in such patients the

drug should be decreased rather than increased for a time. The *second* applies to oedema of *renal origin*; *intravenous injection should be withheld in these* cases until a good diuretic response has been obtained in the first instance by *intramuscular injection.*

Digitalisation.—Although digitalis proves useful in all types of heart failure, it is seen at its best in failure from mitral stenosis with auricular fibrillation.

TABLE XII

Showing the relative value of different methods of premedication in augmenting the natural diuresis produced by mercurial preparations in a series of patients with heart failure.

Order of efficiency	*Method of premedication*	*Diuretic index (average)*
1	Ammonium chloride (30 grains or 4 tablets) two hours before	69
2	Ammonium chloride (15 grains or 2 tablets) three times a day for three days	61
3	Ammonium chloride (15 grains or 2 tablets) three times a day for two days	58
4	Urea (30 grammes) two hours before	57
5	Ammonium chloride (60 grains or 8 tablets) two hours before	54
6	Ammonium chloride (45 grains or 6 tablets) two hours before	53
7	Euphyllin (0 4 grammes) two hours before	49
8	Ammonium chloride (15 grains or 2 tablets) three times a day for one day	39
9	Ammonium chloride (15 grains or 2 tablets) two hours before	39
10	No premedication	34

In such cases digitalis is expected to slow the heart rate and abolish the pulse-deficit (Fig. 232), to induce diuresis and to relieve oedema and breathlessness from lessening the pulmonary congestion Electrocardiographic effects (Fig 233) appear in a day or two. It is immaterial that extrasystoles or coupling of the beats come in the wake of digitalisation provided that the patient is free from nausea, but often the two symptoms are conjoined. The cardiac output is increased by digitalis when the failing heart is enlarged, but in a normal heart digitalis might even diminish cardiac output so that its use in pneumonia, for instance, if not harmful, is not worth while. Whenever it has been decided to use digitalis it needs to be given in proper measure and continuously. By digitalisation is meant that state whereby the dosage of digitalis standard for the patient ensures freedom from

symptoms—symptoms of heart failure on the one hand and toxic symptoms on the other, neither undue breathlessness nor nausea are compatible with a state of satisfactory digitalisation in a patient in whom a favourable digitalis action is anticipated

Rate per minute: 120, 115, 110, 105, 100, 95, 90, 85, 80, 75, 70
Digitalis intake
Days 1 2 3 4 5 6 7 8 9 10
Pulse rate — Ventricular rate

FIG 232—Showing how the pulse deficit is annulled by digitalis therapy

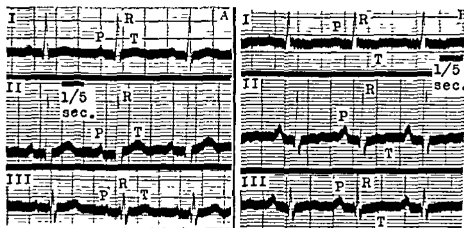


FIG. 233—Electrocardiographic effects of digitalis therapy The tracing in (B) was taken 7 days later than that shown in (A), and after the patient had taken 40 minims of the tincture daily. The R-T segment is depressed in all three limb leads, and the T is diphasic in II and inverted in I.

It is sometimes necessary to induce digitalis-effect quickly (rapid digitalisation) and this is best done by giving 2 or 3 mg. digoxin by mouth or 1 5 mg. intravenously. In this way the action of digitalis is seen within one or two hours (Figs. 234 and 235). For continuous digitalisation the powdered leaf proved to be the best among a number of digitalis preparations when their effects were

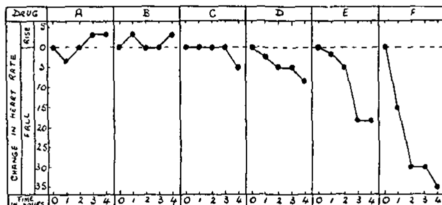


FIG 234—Rapid digitalisation Change in heart rate from various digitalis preparations (A) Digitaline (A & H), $\frac{1}{40}$ gr by mouth (B) Digitalis tincture, 2 drachms by mouth (C) Digitalis leaf, 6 gr by mouth (D) Strophanthin, $\frac{1}{100}$ gr intravenously (E) Digoxin, 1 5 mg by mouth (F) Digoxin, 1 0 mg intravenously.

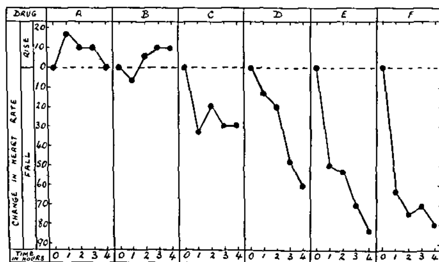


FIG 235—Rapid digitalisation Change in heart rate from different digitalis preparations (A) Lanatoside C, 3 0 mg by mouth (B) Coramine, 1 7 cc intravenously (C) Lanatoside C, 0 8 mg intravenously (D) Strophosid, 1 cc intravenously, (E) Digoxin, 3 0 mg by mouth, (F) Digoxin, 1 5 mg intravenously

TABLE XIII

Heart rate (per minute) in 18 patients with auricular fibrillation, after treatment with different drugs

Patient					*Preparation used*										
Case number	*Age*	*Sex*	*Weight (in lb)*	*No of doses daily*	*Digitalis leaf*	*Digitaline (Nativelle)*	*Digifoline*	*Digoxin*	*Tincture of digitalis*	*Digitaline (A & H)*	*Folinerin*	*Ouabaine*	*Strophanthin*	*Coramine*	*Cardiazol*
1	55	F	106	2	87	88	88	86	96	86	92	82	94	100	108
2	25	F	104	3	65	72	72	70	80	66	96	80	104	86	110
3	56	F	134	3	60	72	72	80	108	80	96	116	92	94	116
4	41	F	98	4	74	84	75	92	78	85	80	134	150	124	128
5	49	F	149	3	98	80	88	98	92	86	110	120	108	134	106
6	45	M	200	3	68	60	74	80	76	76	92	76	88	110	104
7	44	M	184	3	80	84	78	80	110	88	76	84	110	96	98
8	65	F	114	2	64	78	100	98	84	98	90	100	92	152	140
9	48	M	116	2	52	50	56	56	58	64	46	66	74	78	78
10	53	M	147	3	104	115	116	120	88	118	140	108	130	128	135
11	51	M	135	3	70	46	62	66	90	68	104	80	84	114	96
12	58	F	182	2	74	74	92	76	62	84	106	116	106	98	112
13	46	F	84	2	52	62	72	64	72	84	100	124	130	100	130
14	53	F	98	2	80	80	98	88	76	100	72	110	146	90	122
15	37	F	104	3	74	80	78	94	106	94	88	94	90	96	126
16	54	F	154	2	66	102	100	92	70	86	92	88	98	112	120
17	42	F	185	2	84	88	70	77	96	96	98	92	104	104	150
18	59	M	147	2	94	102	120	106	126	110	136	128	120	154	136

compared in patients (Fig. 236). The dosage of digitalis cannot be predicted for any patient and it can only be settled after clinical trial, implying a therapeutic study in every patient. It is not possible to calculate the appropriate dose of any digitalis preparation from biological standardization alone, nor yet from the

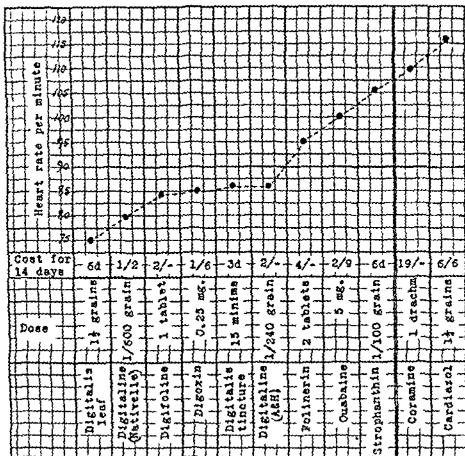


FIG 236.—The effect of drugs on the heart rate in 18 patients with auricular fibrillation An average rate of 90 has been accepted as the uppermost level of satisfactory digitalisation for this series The cost of each drug is quoted for hospital patients receiving it three times a day for a fortnight. Drugs to the left of the upright double line were given over periods of 14 days; those to the right, over periods of 7 days

patient's body-weight. Thus, digitalisation of a patient of heavy weight is sometimes maintained by small doses of digitalis, while that of a patient of light weight might only be reached by large doses The heart rate during successful digitalisation differs widely among patients with auricular fibrillation, so that it might be 115 in one and 52 in another (Table XIII). The heart rate gains the greatest significance when the digitalis effect of different preparations is estimated in the same patient. It has proved a good custom to adopt a standard dose for each digitalis preparation and to vary only the daily dosage. Thus, with powdered

digitalis leaf the single dose of 1 grain should be employed and given once or twice, occasionally three times, and rarely four times a day, according to need. Hallucinations or such mental dysfunction are not uncommon in long-standing failure and their appearance signifies a bad prognosis. Although they are unrelated to treatment, occasionally in failure with normal rhythm, the withdrawal of digitalis has ameliorated the symptoms or caused them to disappear.

Heart failure in specific conditions

Now that the general aspects of heart failure have been described, it remains to discuss the special features of heart failure as met with in different forms of cardiovascular disease. Naturally, the general principles already enunciated apply to the special conditions, but under each heading will have to be mentioned certain facts which are common to these conditions alone or in a large measure.

Hypertensive heart failure —Since hypertension is a common condition, hypertensive heart failure is necessarily a common event. It is probable that this

TABLE XIV

Clinical features which distinguish the two types of hypertensive heart failure.

Clinical feature	*Hypertensive (left heart) failure*	*Hypertensive (right heart) failure*
Previous history of hypertension	Usual	Often missing
Increasing breathlessness on exertion	Present	Present
Nocturnal dyspnoea (cardiac asthma)	Invariable	Less frequent
Cyanosis	None	Common
Enlargement of neck veins	None	Usual
Crepitations at lung bases	None	Present
Enlargement of liver	None	Often present
Ascites	None	Often present
Oedema	None	Present
Tachycardia	Usual	None
Blood pressure	Raised	Usually not raised
Triple rhythm	From the addition of the fourth heart sound	From the addition of the third heart sound
Electrocardiographic changes	Distinctive	Bizarre
Findings on cardioscopy	Enlargement of left ventricle, hilar congestion	Enlargement of left ventricle and right heart; hilar congestion
Prognosis	Better	Worse

clinical entity often goes unrecognized in its entirety for two reasons. First, in hypertensive heart failure showing *left* ventricular failure, heart failure often remains undiagnosed because of the scarcity of the familiar failure signs. Secondly, in hypertensive heart failure showing *right* heart failure, the signs of failure are obvious but its aetiology is often unsuspected because the blood pressure may not then be raised. So unlike are the presenting signs of these two types that they will be discussed separately and their symptoms are arranged for comparison in Table XIV.

Hypertensive (left heart) failure.—The presenting symptom in left ventricular failure is nocturnal dyspnoea. The patient wakes with a start from his sleep and the breathing is rapid, shallow and gasping ; he sits up in bed or he may seek the open window. The air hunger results from the pulmonary venous stasis distending the lung and pleura and initiating a reflex which stimulates the respiratory centre, support for such a theory is gained from the fact that morphine can relieve the attack. The paroxysm of breathlessness often concludes with coughing and the voiding of frothy sputum ; it may not be repeated each night, but as the illness progresses the attacks lengthen, recur most nights, and extra pillows are demanded for an upright posture which provides the greatest immunity from attacks and for this reason the patient may prefer to spend the nights in a chair. The nocturnal posture is important in the differential diagnosis of cardiac asthma from bronchial asthma, in the latter the patient sleeps in the reclining posture or on his side, but in the former several pillows are piled up in an attempt to hold the shoulders forward. The other signs of hypertensive (left heart) failure have been arranged in Table XIV where they are compared with those found in hypertensive (right heart) failure. The signs familiarly identified with failure, namely, cyanosis, distension of veins in the neck, enlargement of liver, ascites and oedema, are missing. Even the lung bases are clear to auscultation during the early months, although obvious hilar congestion is already present on cardioscopy. Naturally, when left ventricular failure has persisted for some time oedema does appear, but during the first months of the illness scarcity of symptoms mars the early diagnosis of the condition. It needs to be emphasized again that the onset of nocturnal dyspnoea in a patient with enlargement of the left ventricle from any cause, notably hypertension, indicates left ventricular failure, that triple rhythm from the addition of the fourth heart sound is an invariable auscultatory sign, and that pulmonary congestion will show on cardioscopy.

The *electrocardiogram* demonstrates the higher grade of left ventricular preponderance so that the T wave is inverted in lead I, with depression of the S-T segment, in addition to a deep S_3 and S_2 signifying left axis deviation. In lead IVR the T may be upright and this by itself may exclude a diagnosis of anterior cardiac infarction, it is when the T wave in this lead is inverted that the diagnosis becomes confused. In this circumstance, help is obtained from the chest lead CR_7. Thus, if the T inversion in CR_7 is greater than in IVR hypertensive effects are uppermost, although the addition of cardiac infarction cannot be excluded with certainty, especially the antero-lateral kind towards the apex which itself produces inversion of the T wave in leads I and CR_7.

On *cardioscopy* the left ventricle is found to be enlarged and the presence of pulmonary congestion is confirmed by finding increased density of the vessels at

the hila As the condition advances some degree of enlargement of the right heart becomes added, and so may pleural effusion.

Hypertensive (right heart) failure.—Allusion has already been made to the development of signs of right heart failure when left heart failure has persisted for some months, but attention needs to be directed specially to a large group of patients with hypertensive heart failure in whom the blood pressure is not raised at the time of examination In the medical history of such patients, too, it is uncommon

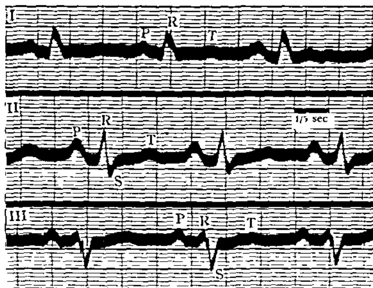


FIG. 237—Right heart failure in hypertension with a normal blood pressure value

to obtain information that the blood pressure had been found raised in the past. Cardiac asthma may be present, but shortness of breath on exercise is a more prominent feature of this group On clinical examination the signs of right heart failure are uppermost, so that cyanosis, distension of veins in the neck, crepitations at the lung bases (often with hydrothorax), slight distension of the liver (sometimes with ascites), and oedema, are present. The blood pressure may be 150/90, but the apex beat is near the anterior axillary line, telling of great cardiac enlargement. The heart rate is slower than in the previous group, and triple rhythm, although not always as distinct, is present from addition of the third heart sound ; this auscultatory sign should be sought for diligently because it provides material help in the diagnosis. Before allocating a patient to this group it is necessary to exclude two other conditions, namely, heart failure from cardiac infarction or from aortic stenosis.

The *electrocardiogram* is not so distinctive as in the patients showing left ventricular failure, but it is never normal. Such a tracing is shown in Fig 237.

On *cardioscopy*, in addition to enlargement of the left ventricle and hilar congestion, which are also characteristic findings in left ventricular failure, there is



Fig 238—*Exaggeration of* left auricular impression (1) in the right oblique position from left ventricular enlargement in hypertension The elongated and tortuous descending aorta (2) *produces* a cascade-like effect on the barium stream.



Fig 239—The enlarged left ventricle (1) from hypertension has caused the *left auricle to impress* the oesophagus in a characteristic way, producing a claw-like curve (2) Aortic arch impression (3) Left bronchus impression (4). Right ventricle (5)

much enlargement of the right auricle, and of the right ventricle as well. These added changes give to the cardiac silhouette the appearance found in mitral stenosis with prominent cardiac enlargement. The characteristic curve of left auricular enlargement of mitral stenosis in the right oblique position is closely simulated (Fig. 238), but examination in the left oblique position demonstrates that this accentuation of the left auricular impression is not due to primary enlargement of the left auricle as in mitral stenosis, but is the result of displacement of the left auricle by the enlarged left ventricle (Figs. 239 and 240).

Treatment

The rules outlined for the general management of established heart failure apply equally to hypertensive heart failure, so that rest in bed or reduced activities during the ambulatory phase, a suitable device to maintain the upright posture at night, and a diminished fluid and sodium intake are important accessories, but the organized

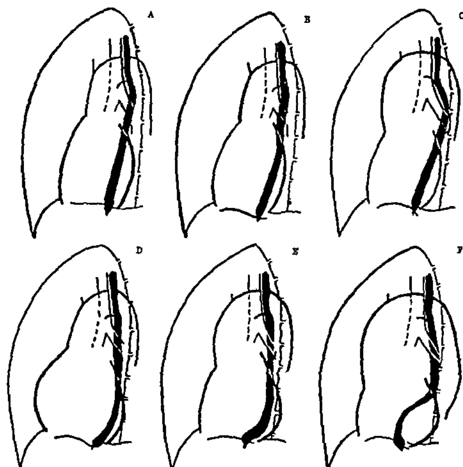


Fig. 240.—Variation in the left auricular impression in the left oblique position (A) Plumb-line in health. (B) Comma curve in health and in mitral stenosis. (C) Effect of aortic elongation without cardiac enlargement (D) Crescent curve in mitral stenosis and left ventricular enlargement. (E) Sickle curve in left ventricular enlargement. (F) Claw curve in left ventricular enlargement with aortic elongation



FIG 241 —Hypertensive heart failure. Gross pulmonary congestion from pulmonary oedema (1) in (A) is absent in (B), taken after one month in bed and after four injections of a mercurial diuretic Left ventricle (2) is enlarged. Ascending aorta (3) and descending aorta (4) prominent from aortic elongation

use of mercurial diuretics must take pride of place in the treatment of hypertensive heart failure. Not only must they be used in the variety of right heart failure in which oedema is an outward sign of fluid retention, but they should be given in early hypertensive (left heart) failure in which there is no obvious and superficial evidence of fluid retention. Indeed, in such patients, in whom the presence of

nocturnal dyspnoea and pulmonary congestion (visible on cardioscopy) are proof of hidden fluid retention, mercurial diuretics are seen at their best (Fig. 241). Nocturnal dyspnoea may disappear after some two or three injections, and the patient may even continue with his customary occupation provided it does not entail exertion. Cardioscopy conducted at intervals during this period serves as a means of assessing the progress of a case as judged by the extent to which congestion of the hilar vessels recedes.

Digitalis has value in the treatment of hypertensive heart failure, and it is good practice to give 1 grain of the leaf twice each day and later once daily.

Morphine should be given each night at the start and withdrawn when successful diuresis has been induced on two occasions. Although aminophyllin may produce

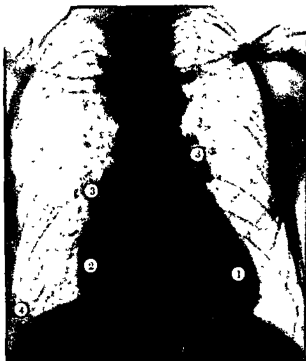


FIG 242.—Thyrogenic heart failure Slight enlargement of the left ventricle (1) and greater of the right auricle (2) Much hilar congestion (3) and right hydrothorax (4). Auricular fibrillation was present and triple heart rhythm from addition of the third heart sound.

benefit in cardiac asthma it is unlikely to equal the success obtained by digitalis and mercurial diuretics following the preliminary administration of morphine for a night or two.

Although the response to a well-devised drug treatment in hypertensive heart failure is often very satisfactory, and sometimes even dramatic at the start, the unfavourable prognosis identified with the condition means that the initial benefit cannot last, and sooner or later the diuretic effect of the mercurial salt wanes; whenever this change is noticed a cautious view of the immediate prognosis is justified, and the drug should be reduced in such circumstance and not increased.

Heart failure in mitral stenosis.—During early heart failure in mitral stenosis the rhythm may remain normal. In this circumstance, if breathlessness is present on exercise and hilar congestion is noticeable on cardioscopy, and if digitalis does not bring about the improvement expected of it, the use of a mercurial diuretic is indicated. When auricular fibrillation has set in, digitalis therapy is seen at its best. If, in the course of adequate digitalisation, oedema persists, it is necessary to apply mercurial diuretics as well. Should heart failure in mitral stenosis be precipitated abruptly with auricular fibrillation at a high rate, digitalisation may be induced quickly.

Thyrogenic heart failure.—The heart rhythm seldom remains normal when failure has appeared in thyroid toxaemia, so that auricular fibrillation is common, and less frequently auricular tachycardia occurs. On the other hand, paroxysmal auricular fibrillation is often present without failure. In thyrogenic failure clinical evidence of cardiac enlargement may be wanting, but some degree of this, small as a rule, is always seen on cardioscopy. The presence of the third heart sound in thyroid toxaemia prevsages both cardiac enlargement and failure (Fig 242), so that it is a valuable sign.

Subtotal thyroidectomy should cure the condition, but preparatory treatment with iodine, digitalis, and sometimes with mercurial diuretics, is necessary. As a rule, digitalis can be withdrawn on the fifth day after operation and quinidine should be given within a fortnight if normal rhythm has not been reinstated by that time. No good purpose is served by trying to establish normal rhythm before the operation.

Heart failure in aortic incompetence and aortic stenosis.—Aortic incompetence from syphilitic endocarditis or aortitis usually leads to heart failure with normal rhythm, and so does the rheumatic lesion if the mitral valve is less severely damaged than the aortic valve. Similarly, with aortic stenosis the rhythm usually remains regular. In such cases, when heart failure sets in, the prognosis becomes ominous and, in the treatment, mercurial diuretics need to be used along with digitalis.

Heart failure in emphysema.—The severe breathlessness associated with emphysema is usually an expression of a respiratory embarrassment and the direct outcome of the loss of elasticity of the alveolar walls; it is seldom the result of heart failure. Failure, however, does take place sometimes and in this event there will be present a triple rhythm from addition of the third heart sound, moderate enlargement of the right auricle, right ventricle and pulmonary artery on cardioscopy, and in the electrocardiogram there may be a deep S wave in lead I and an inverted T wave in leads II, III and CR_1. These special signs are conjoined with the usual evidence of right heart failure, and the heart rhythm remains regular. Prognosis is serious. In this group of patients, too, digitalis therapy has to be reinforced with mercurial diuretics, but the results are necessarily oft-times disappointing because of the serious nature of the condition.

Heart failure in congenital heart disease.—In this instance, too, the signs of right heart failure are present with normal rhythm, and, for a time, mercurial diuretics give good results in treatment. In the case of patent ductus arteriosus it is better to prevent heart failure by ligation of the ductus before the onset of such

symptoms A warning is necessary not to mistake the breathlessness which accompanies the cyanosis common to congenital heart disease presenting a venous-arterial shunt for the breathlessness of heart failure.

Heart failure in cardiac infarction.—Although arrhythmia especially auricular fibrillation, auricular tachycardia and heart block, is sometimes present in cardiac infarction showing heart failure, *normal rhythm is preserved in the majority of* cases. Failure may be an early event and be ushered in abruptly at the start, especially in the presence of hypertension. In the absence of hypertension, cardiac enlargement is minimal in heart failure following cardiac infarction (Fig 243). As a late manifestation in cardiac infarction, too, failure may be conjoined with



FIG. 243 —Heart failure in cardiac infarction There is only slight enlargement of the heart Pulmonary congestion (1)

hypertension, and mercurial diuretics together with a resting period form the standard method of treatment Digitalis need not be withheld on the supposition that it might precipitate ventricular fibrillation Occasionally cardiac aneurysm is a feature of heart failure developing in long-standing cardiac infarction.

Heart failure in nephritis—Heart failure is a common event in nephritis whenever *it is associated with hypertension, and this also applies, although less com*monly, to acute nephritis A more general acceptance of the common incidence of heart failure in nephritic hypertension will ensure a wider application, in these cases, of measures directed to relieve cardiac rather than renal failure. Naturally it is necessary to exercise caution in the use of mercurial diuretics when heart failure in hypertension is associated with acute nephritis. If the decision has been made to use them, and the need to do this will often arise, it is better to give the mercurial

salt intramuscularly; it should not be given intravenously unless good diuresis has been obtained first by intramuscular injection.

Nephritis is sometimes a sequel of heart failure, but it is seldom uppermost in the clinical picture in that event.

Heart failure in constrictive pericarditis.—Not all cases of adherent pericardium develop signs of heart failure and even then breathlessness is uncommon at the start because pulmonary congestion is usually slight The picture of failure is also modified in this condition through the absence of cardiac enlargement. When symptoms are ushered in abruptly in adherent pericardium it is necessary first to observe the effects of rest and mercurial diuretics. Should it be necessary to combat fluid-retention by the continuous use of mercurial salts after two months, constrictive pericarditis may be presumed present, and cardiac decompression by pericardiectomy should be advised, because the constrictive effects will last without such surgical procedure. Such treatment should be carried out early in the illness and before congestion fibrosis of the liver becomes severe

Heart failure in complete heart block.—When complete heart block is complicated by heart failure, digitalisation should be tried in addition to the regular use of mercurial diuretics, even though Stokes-Adams attacks are present. It is possible to add the benefit of digitalisation in complete heart block because the ventricular rate is seldom slowed by digitalis when idioventricular rhythm is present

Heart failure in arterio-venous aneurysm.—When heart failure symptoms, enlargement of the heart, and a collapsing pulse, take place in the absence of an aortic diastolic murmur, an arterio-venous fistula of some size can be presumed to be present. Treatment should consist in removing it, if accessible, after adequate pre-operative treatment by rest, mercurial diuretics, and digitalis.

CHAPTER 13

HEART DISEASE AND PREGNANCY

NOTHING is gained by discussing separately every form of heart disease in relation to pregnancy Naturally, mitral stenosis will be the commonest lesion, but the principles that govern treatment of the patient during pregnancy are essentially the same in the case of the rarer cardiac abnormalities. It is necessary, on the other hand, to inquire about the influence of pregnancy in precipitating heart failure, and to pay special attention to the need of modifying the management of pregnancy and labour in heart disease both with and without heart failure. There is not any evidence that pregnancy has any effect on the progress of the pathological lesion in rheumatic, syphilitic, or hypertensive heart disease. Moreover, it is unlikely that pregnancy by itself can induce failure in a patient with heart disease when failure signs are absent at the commencement of cyesis and when the severity of the heart lesion is not such as to give rise to failure in a short time in the absence of pregnancy. When failure is already present, however, cyesis can aggravate the symptoms The least favourable aspect of pregnancy in relation to heart disease is found in the added domestic burden and diminished opportunity for resting, which successive births inevitably bring to the mother.

The management of pregnancy in a patient with heart disease may be discussed in relation to five phases, namely, the pregestation period, the first three months of pregnancy, the later months of pregnancy, parturition and the post-natal period.

PREGESTATION PERIOD

If heart disease is known to be present the advice of the medical attendant may often be sought concerning the dangers of child bearing. Should symptoms of heart failure be absent it is rare for them to be ushered in during the first pregnancy, although the patient should remain under medical supervision until the birth. If symptoms and obvious signs of failure are already present it should be made clear that pregnancy is a very undesirable state. When failure signs are less obvious, the patient seeking this advice does not, as a rule, readily admit to breathlessness, so that evidence of failure must be looked for on cardioscopy as well as on clinical examination When pulmonary congestion is slight and both partners of the marriage are anxious that there should be a child, they may be told that with adequate medical supervision and treatment the pregnancy and labour should be satisfactory. Sterilization during the immediate post-natal period should be advocated in such a case, but the decision on such a procedure should always rest with the patient and her husband after paying due regard to medical advice.

FIRST THREE MONTHS OF PREGNANCY

The decision to be taken during this period concerns therapeutic abortion Putting aside the wishes of the patient this is not difficult. Thus, if failure signs are absent, pregnancy may proceed naturally to term, but should cardioscopy show heavy hilar shadows, therapeutic abortion followed by sterilization is the best course

to follow. If failure signs are early, and the hilar congestion is slight, a discontinuation of pregnancy is never justified in the primipara, nor is it in a second pregnancy if the parents are anxious that it should proceed to term. If the patient wishes to continue with the pregnancy in the face of advice to the contrary, she must adhere to the rules governing any case of heart failure, namely, to rest adequately, to restrict the intake of fluid and common salt, and to take digitalis continuously, especially in the presence of auricular fibrillation, which is naturally uncommon in patients with mitral stenosis during child-bearing age, even mercurial diuretics may have to be used. Sterilization should take place after parturition in such cases.

LATER MONTHS OF PREGNANCY

For a patient with heart disease, either with or without failure, a measure of rest must be assured during the last months of pregnancy, while admission to hospital should take place if failure is present, at least three weeks before the birth is due.

If there are no obvious clinical signs of failure, and if pulmonary congestion on cardioscopy is minimal, the pregnancy may be allowed to proceed to full term In these circumstances early induction of labour is not indicated, nor is Caesarean section, unless on other grounds.

When heart failure has appeared, treatment should be directed primarily to its relief When the patient has already attended for supervision during the ante-natal period, the treatment of failure will have already been organized, but in the untreated case, presenting for the first time as an emergency, alleviation of severe failure symptoms will cause anxiety, in such cases auricular fibrillation is more common and rapid digitalisation by digoxin should be the first procedure followed later by continued digitalisation. If there is much fluid retention, mercurial diuretics need to be given as well Furthermore, when right-sided congestive heart failure is associated with conspicuous cyanosis, venesection is good treatment. Only when clinical improvement has followed these measures will attention be turned to the pregnancy. In a primipara Caesarean section is often the best procedure to adopt, but in a multipara when failure symptoms are abating the pregnancy may be allowed to proceed to full term, or otherwise terminated earlier by induction methods. It is amongst such emergency cases that the mortality rate is high Rest has been inadequate and no specific treatment of the heart failure has taken place, so that when first seen the patient is desperately ill with the symptoms common to abrupt heart failure. In such a patient also, even when the symptoms have been temporarily relieved and the pregnancy terminated, death may take place from the sudden re-entry of symptoms of severe failure.

MANAGEMENT OF PARTURITION

In a primipara without obvious failure signs, or a multipara with moderate failure, pregnancy may be allowed to proceed to full term and the second stage of labour should be expedited with the use of forceps. Caesarean section is desirable in many primiparous patients who show moderately severe failure, whereas the same clinical state in a multipara would indicate the need for an early induction of labour and a shortening of the second stage. Indeed the obstetrician's aim in all such cases will be to shorten the process of labour, and at the same time ensure the least amount of disturbance to the mother.

In regard to anaesthesia there is only need to emphasize that the efficiency with which anaesthesia is induced matters much more than the kind of anaesthetic employed. Open anaesthesia with ether and chloroform, or with ether and gas and oxygen are equally satisfactory. Cyclopropane might be the anæsthetic of choice, but it can only be administered by those skilled in its use.

POST-NATAL PERIOD

Supervision of the mother should not cease at the birth of the child. The load on a mother's damaged heart is increased in greater measure from the addition of physical exertion inseparable from the advent of a baby in the home, than from any upset of the circulation occasioned by the foetus in the womb. If the onset of failure is to be postponed or its symptoms, if already present, are to be halted, adequate help must be provided in the home to give the mother the opportunity to rest. Such social service should hold equal place with continued medical supervision and digitalisation.

It needs final emphasis that the foregoing remarks should not be regarded as rigid rules applied to every case of pregnancy in heart disease, but they are meant to be guiding principles in deciding upon the course to follow in the treatment of

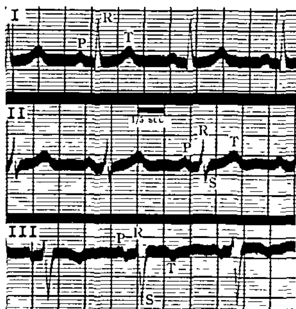


FIG 244 —Raised diaphragm from cyesis. The P, QRS, and T waves are inverted in lead III.

individual cases. It must be understood that circumstances peculiar to each patient should finally determine the plan of treatment, and that the first and successive examinations should include cardioscopy.

Concerning *prognosis* it may be stated in general terms that the morbidity and mortality rates connected with heart disease during the pre-natal and immediate post-natal periods are lowest when a precise diagnosis of the heart lesion has been

made early, when the onset or progress of heart failure has been watched by cardioscopy, and when supervision has been applied from the commencement of pregnancy, under the dual control of the physician and obstetrician. Such ideal circumstances should be the aim of any department organized to deal with these cases.

A warning is also necessary about the interpretation of certain electrocardiographic and cardioscopic signs which are the natural outcome of an elevated diaphragm in cyesis. Thus, in the *electrocardiogram* the three primary waves in lead III may be inverted (Fig 244). Again, on *cardioscopy* the heart shadow may appear squat in the anterior view, and in the right oblique position with barium in the oesophagus the natural left auricular impression is accentuated, such findings should not be interpreted as evidence of mitral stenosis, but should be recognized as the effects of a raised diaphragm.

CHAPTER 14

THE HEART IN ENDOCRINE DISORDERS

THYROID DISEASE

THYROID TOXAEMIA

WHEN a goitre has produced toxic effects over a long period, especially in elderly subjects, changes in the cardiovascular system are usually evident on clinical and radiological examination Some of the physical signs are an outcome of dilatation of the capillaries which form the lesser vascular beds, and others appear to be the result of a toxic action on the heart and its controlling nervous mechanism. The differential diagnosis between thyroid toxaemia and neurosis in the presence of an innocent goitre, is often difficult, but attention to thyrotoxic effects on the cardiovascular system will help to decide, and herein lies the chief importance of the subject These effects will now be discussed

Sinus tachycardia

Thyroid toxaemia is not to be regarded as operating unless tachycardia is present; exceptionally the higher rate is halved by sino-auricular block affecting alternate beats, treatment with iodine or thiouracil may also account for the slower rate



FIG 245.—Extrasystoles in the presence of tachycardia (about 100 a minute) Every third beat is premature Healthy female aged 17 years

in a given case. The tachycardia persists during sleep, increases from exertion or excitement, and subsides only slightly on resting, but it subsides more especially after medicinal treatment.

Extrasystoles

When extrasystoles are infrequent they usually disappear after induced tachycardia. Should occasional extrasystoles appear in the presence of tachycardia, thyroid toxaemia should be sought as the explanation, although often it may prove not to be the cause (Fig. 245).

Auricular fibrillation

Auricular fibrillation is a common event in elderly patients in whom thyrotoxicosis is severe and has persisted for some time. In the several series of cases published the incidence of the arrhythmia has varied from 10 to 20 per cent. Although the heart is enlarged whenever fibrillation has been established, the enlargement is never so great as that associated with fibrillation in mitral stenosis or in hypertension. The heart rate in thyrogenic fibrillation can be slowed by iodine as well as by digitalis although not as readily. It can often be abolished temporarily by quinidine, and permanently by subtotal thyroidectomy in over 70 per cent of patients. In cases needing digitalisation before operation the drug should be discontinued some four days after the operation in case it should impede a spontaneous reversal to normal rhythm, this event should be awaited over a period of ten days before quinidine therapy is started. Quinidine effects a return to normal rhythm in more than one-half of the cases in which fibrillation has persisted after the operation; the other half is uninfluenced by quinidine, but the patients remain free from symptoms if the heart rate is kept low with light digitalisation

When a patient is subject to repeated brief attacks of auricular fibrillation, goitre is often the cause, for in other conditions causing fibrillation the arrhythmia is more frequently established from the start. In these circumstances the goitre presents as an adenoma in an elderly subject and the attacks of auricular fibrillation may be the only noticeable toxic effect apart from loss of weight. Removal of the thyroid swelling in these cases frequently prevents the recurrence of further fibrillation.

Auricular tachycardia

When the thyrotoxic effects are severe either before operation or during the post-operative crisis, sinus tachycardia may give way to auricular tachycardia As a rule the onset of this form of arrhythmia is an ominous sign. Iodine, quinidine, and digitalis, should be given a trial in treatment. Thiouracil is also on trial in such a circumstance and is likely to prove valuable.

Collapsing pulse

A water-hammer pulse is a common feature of thyroid toxaemia; the pulse pressure is increased owing to a slight rise in the systolic blood pressure and a greater fall in the diastolic pressure The physical sign disappears after a successful thyroidectomy.

Hypertension

A raising of both the systolic and the diastolic blood pressures may occur in patients with thyroid toxaemia. That this is a true thyrogenic hypertension is doubtful, because subtotal thyroidectomy which removes the other symptoms common to

the illness, does not reduce the blood pressure, although this does not exclude the belief that thyroid toxicity in the first instance initiated a state of hypertension which might be maintained by some other agency. Anyhow, the incidence of hypertension in thyroid toxaemia is common enough to suggest its thyrogenic origin.

Cardiac enlargement

In the absence of associated hypertension, it is rare to find clinical evidence of cardiac enlargement in thyrotoxicosis even though auricular fibrillation has been present for some time Less obvious enlargement, however, can frequently be made out on cardioscopy In about one-half the patients in whom the illness has been present for about two years, a degree of cardiac enlargement can be made

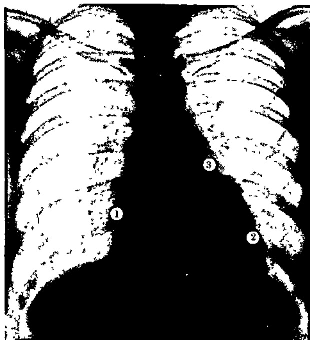


FIG 246—Thyroid toxaemia in a female aged 30 years producing slight enlargement of the right auricle (1) and left ventricle (2), and prominence of the pulmonary arc (3)

out When viewed in the anterior position the slight enlargement involves the right auricle and left ventricle, and when these changes are associated with a prominence of the pulmonary arc a characteristic ham-shaped cardiac silhouette is presented (Fig. 246) In cases where fibrillation has been present for some time, examination in the right oblique position with barium in the oesophagus may show selective enlargement of the left auricle (Fig. 247) No noticeable diminution of the enlarged cardiac silhouette has been observed after an operation which has successfully removed all toxic effects.



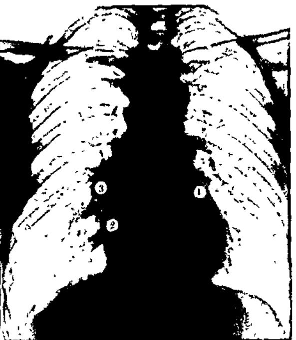


FIG 248.—Thyrogenic auricular fibrillation. Slight enlargement of conus (1), and of right auricle (2). Hilar congestion (3)

FIG 247.—Thyrogenic auricular fibrillation. Enlargement of conus (1), and of left auricle (2) which produces a deep impression on the oesophagus

Heart failure

The common symptoms of heart failure in thyroid toxaemia are associated with only moderate enlargement of the heart as seen on cardioscopy which also shows pulmonary congestion (Fig. 248) Clinically the pulse is usually irregular from auricular fibrillation, but the sign which predicts the presence of right heart enlargement and pulmonary congestion on cardioscopy, is the finding of triple rhythm from the addition of the third heart sound. A low T wave in lead II of the electrocardiogram, and an inverted T in leads III and CR_1, are other expressions of right heart preponderance.

Myxoedema

Only a proportion of cases of myxoedema shows changes in the heart; even when they are present, clinical examination by itself usually fails to detect them.

Pathology—Owing to the paucity of pathological studies of the heart muscle in myxoedema, no one explanation of the actual cause of the cardiac changes can be given. The heart cavities appear to be dilated, and there may be an alteration in the water content of the myocardium, but its infiltration with myxoedematous tissue cannot be demonstrated

Symptoms and signs—It is rare for symptoms to arise which can be attributed directly to the heart Thus, shortness of breath and oedema of the extremities can be the direct outcome of myxoedema which is often associated with moderate or even severe anaemia. Bradycardia is usual and the pulse is small unless there is incidental hypertension. The apex beat is quiet and its slight outward displacement is difficult to elicit on clinical examination

On *cardioscopy* considerable enlargement of the heart shadow may be present, and this effect is exaggerated by a raised diaphragm which is a common feature of most cases The heart shows little movement; and this sign along with the increase in the cardiac area gives it the appearance of pericardial effusion; the outline may also be stencilled as in the case of pericardial effusion, but on account of the bradycardia Treatment with thyroid extract quickly diminishes the size of the heart shadow (Figs 249 and 250)

The *electrocardiogram*, too, is distinctive in myxoedema. Characteristically it shows sinus bradycardia, low amplitude of all the waves, and flattening or inversion of the T waves. These changes are corrected after a short period of thyroid therapy (Fig 251)

Retrosternal goitre

A retrosternal goitre may cause enlargement of the heart like that in the suprasternal variety, depending on the degree of its toxic effects, but it is in connexion with the appearances on cardioscopy that the condition is described here. Sometimes in elderly subjects the enlarged thyroid cannot be felt in the neck, and when obvious wasting is found on clinical examination of such patients, a retrosternal goitre should be sought for diligently on cardioscopy, because it might not present easily in the anterior view. The position of choice is the left oblique in which two effects must be looked for; one is a displacement by the goitre of the trachea to the right, when the trachea is sometimes constricted, but of



Fig 249.—Myxoedema Enlargement of the heart (A) disappeared after two months on thyroid treatment (B) The height of the diaphragm is the same in both teleradiograms



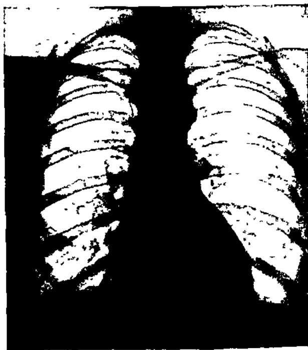


FIG 250 — Myxoedema Enlargement of the heart (A) was absent in (B) after three months on thyroid treatment Female aged 43 years

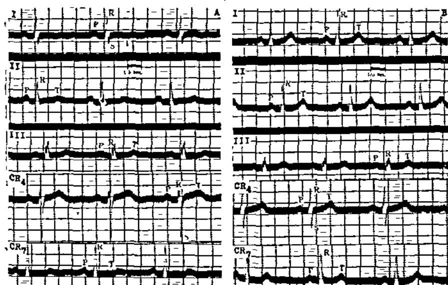


FIG 251 —Myxoedema Sinus bradycardia and low waves in (A) returned to normal (B) after thyroid therapy



FIG 252 —Extension of retrosternal goitre (1) to trespass on the aortic triangle (2), and depressing the aortic arch (3) Left pulmonary artery (4)

greater value in diagnosis is the trespass of the gland on the aortic triangle (Fig. 252). In some instances this radiological landmark is obliterated by the goitre which also depresses the aortic arch, forming the base of the triangle. In addition to diagnosis, this sign delineates for the surgeon the size of the gland which he has to extricate.

SUPRARENAL DISEASE

ADDISON'S DISEASE

Destruction of the suprarenal cortex, and to a lesser extent the medulla, by tuberculosis or simple atrophy, produces cardiovascular effects of which hypotension is a characteristic one. It is likely that in part this is a pituitary effect for the number of the basophil cells in the anterior lobe is greatly reduced.

The *electrocardiogram* may show conspicuous irregularities in the form of inversion of the T waves, but more often it is a normal tracing. The abnormal

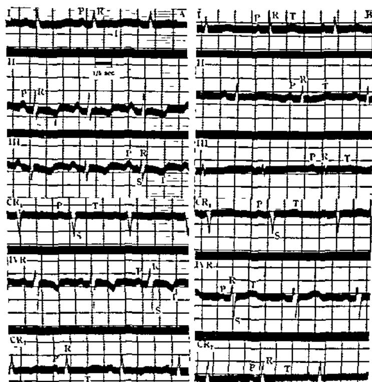
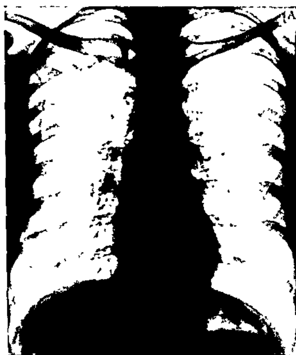


FIG 253 —Addison's disease. Inversion of the T waves in (A) taken before therapy, corrected in (B) after injections of desoxycorticosterone acetate and the administration of common salt. Such changes were associated with a change in the size of the heart (Fig 254 (A) and (B)), but were unconnected with the potassium value

cardiogram is not related to the serum potassium value, but is more closely associated with the size of the heart, thus, it was abnormal in one patient when the heart was small, and it returned to normal when the heart enlarged after treatment with desoxycorticosterone acetate and common salt (Fig. 253).



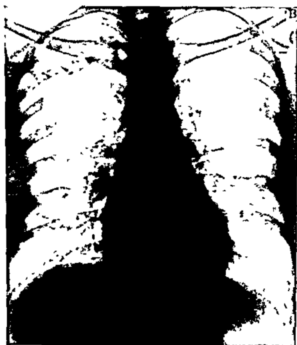


FIG 254.—Addison's disease in female aged 43 years Change in size of heart in one month following daily injection of 5 mg of desoxycorticosterone acetate and 2 5 gramme of common salt by mouth. (A) was taken before treatment and (B) after treatment No change in blood pressure during the same period



FIG 255—Addison's disease in female aged 34 years Change in size of heart and onset of pulmonary congestion in three months after daily injection of 10 mg desoxycorticosterone acetate and 10 gramme of common salt by mouth No change in blood pressure during this period. Progress of therapy was not controlled by successive teleradiograms of the heart, the one marked (A) was taken in the search for pulmonary tuberculosis before treatment started, and the other marked (B) was taken three days before death because physical signs had developed at the right base



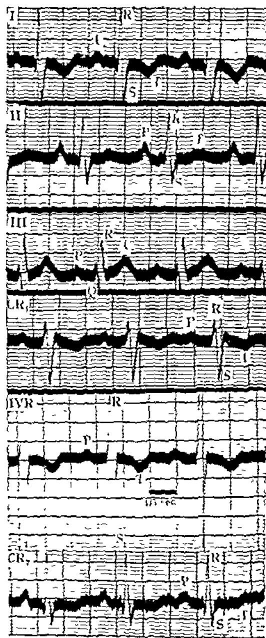


Fig 256.—Suprarenal tumour The T wave is inverted in leads I, CR_1, CR_4 and less in CR_7

On *cardioscopy* the heart in some cases appears to be normal in size, but in others it is small (Fig 254) due to the loss of cellular glycogen, potassium and water The heart enlarges as the clinical condition improves from treatment with desoxycorticosterone acetate Since the blood pressure in many patients is not appreciably altered during successful therapy, the size of the cardiac silhouette as determined by cardioscopy is a reliable index of overdosage of salt (Fig. 255).

SUPRARENAL TUMOUR

Chromaffin tissue tumour of the adrenal medulla (phaeochromocytoma) may give rise to characteristic symptoms which may appear as episodes. They consist of palpitation, nausea, dizziness, a rise in blood pressure, cold and pale extremities, and acute pulmonary oedema.

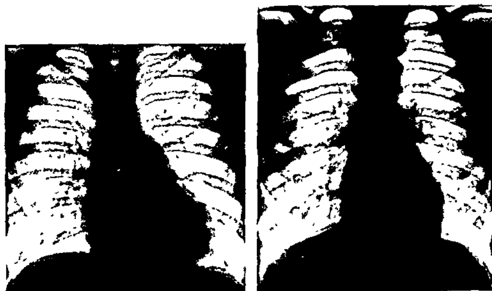


FIG 257—Gigantism Cardiomegaly in a boy aged 16 years whose height was 6 feet 2 inches is shown in (A) (B) is from a healthy boy of 16 years for comparison

Some enlargement of the left ventricle is apparent on *cardioscopy* in such cases, even in the intervals when the blood pressure shows a normal value. The *electrocardiogram* (Fig 256) is a valuable test for it may suggest the diagnosis when other clinical signs are equivocal The common change is an inversion of the T wave in one or more leads, and this may take place in the absence of maintained hypertension.

PITUITARY DISEASE

Destruction of the anterior lobe of the pituitary gland in *Simmonds disease* is associated with a low blood pressure, a small heart and inverted T waves in the electrocardiogram

In *eosinophilic tumours* of the anterior lobe giving rise to acromegaly after the age of puberty it is rare to find cardiomegaly, but gigantism in younger subjects shows it more often (Fig 257)

In *basophilism* a hyaline change in the basophil cells is evidence of their increased activity, and this results in hypertension producing its own characteristic cardioscopic and cardiographic effects. The hypertension is of the papilloedemic type. Pituitary basophilism is also associated with certain thymic and adrenal tumours

CHAPTER 15

THE HEART IN MISCELLANEOUS CONDITIONS

Friedreich disease

It has been the custom to regard Friedreich disease as affecting only the central nervous system, but it is now known that the heart is also often affected Sometimes the cardiac symptoms are uppermost in the clinical picture.

Pathology—The changes in the heart may be localized or widespread They consist of thickening of the muscle fibres and diffuse fibrosis with corresponding enlargement of the heart Microscopical examination demonstrates fatty degeneration of the muscle fibres with slight chronic inflammatory infiltration and fibrosis.

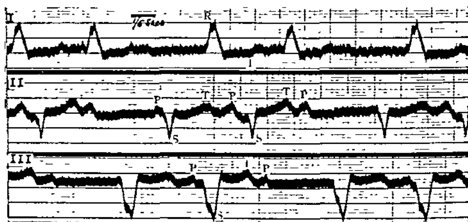


Fig 258—Complete block and left bundle branch block with extrasystoles in a female aged 22, with Friedreich disease, who died in a Stokes-Adams attack A brother had died in similar circumstances at the age of 18 years

Symptoms and signs—Stokes-Adams attacks from complete heart block and the symptoms of heart failure are the common clinical manifestations of heart involvement, and sometimes paroxysmal tachycardia.

The *electrocardiogram* is abnormal in about two-thirds of the cases and in one-third the changes are conspicuous When Stokes-Adams disease is added in Friedreich disease the cardiogram shows complete heart block (Fig 258), and this indicates a serious prognosis. Other cardiograms of Friedreich disease show a coronary type of curve of the T_1 variety (Fig 259) or the T_3 type (Fig 260), and others may show T wave inversion in all limb leads (Fig. 261).

The abnormal nervous system signs are more widespread in patients showing the more conspicuous cardiographic changes. Of greater significance is the higher incidence of a family history of Friedreich disease in those with an abnormal



Fig 259 —Friedreich disease in a male aged 22 years, producing a T_1 type of coronary curve; the T wave was also inverted in the chest lead CR_4



Fig 260 —Friedreich disease in a male aged 20 years, producing a T_3 type of coronary curve; the T wave was also inverted in CR_4 and CR_7.

cardiogram, and the tendency of members of the same family to show identical cardiographic changes. The electrocardiogram may help to establish the diagnosis of Friedreich disease when the neurological manifestations are not altogether typical of the condition; an abnormal tracing lends support to the diagnosis, but a normal curve does not exclude it. Some degree of cardiac enlargement is common in those cases showing heart block (Fig. 262).

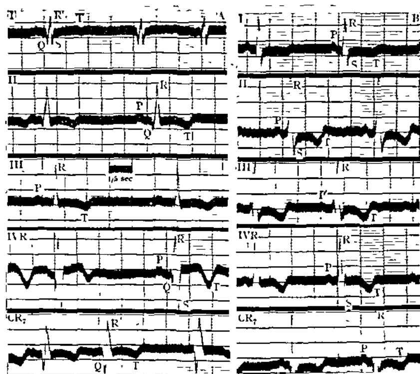


FIG. 261.—T wave inversion in all three limb leads and in IVR and CR_7 in Friedreich disease. (A) is from a male aged 26 years, and (B) from a sister aged 32 years.

Both as regards diagnosis and prognosis, the investigation of a patient with Friedreich disease is incomplete without electrocardiography and cardioscopy.

Myotonia atrophica

The medical history of patients with myotonia atrophica has shown the need to implement the myopathic signs with others which might lead to an earlier diagnosis and spare the patients the unfair judgment passed on them by employers and doctors alike in regarding them as nervous subjects or even malingerers. Signs discovered during the examination of the cardiovascular system contribute to the surer and earlier diagnosis of the condition.

Symptoms and signs.—The pulse is often small and occasionally slow. The blood pressure is sometimes low. The first heart sound in the mitral area commonly shows splitting, and triple rhythm may appear if the P–R period is sufficiently elongated.



Fig 262 —Friedreich disease Generalized enlargement of the heart in a male aged 40 years, with complete heart block as a complication of the disease

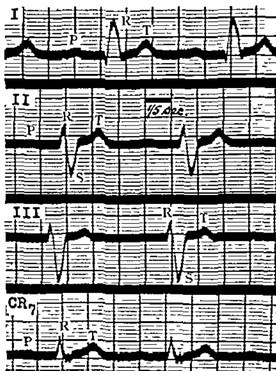


Fig 263 — Myotonia atrophica Prolonged P–R period and left axis deviation Although the QRS complexes are wide the T is upright *in leads I and CR_7, so* that it differs from the tracing of bundle branch block

The changes which commonly characterize the *electrocardiogram* include elongation of the P–R period, low voltage of the P wave, slurring of the QRS complex and left axis deviation (Fig. 263); occasionally there is A–V dissociation and in such patients there may be Stokes-Adams attacks (Fig 264)



FIG 264—Myotonia atrophica 2 to 1 heart block.

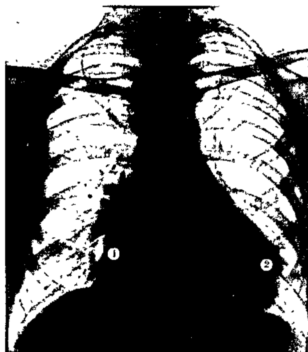


FIG. 265—Myotonia atrophica. Generalized enlargement of the heart in a male aged 34 years whose electrocardiogram showed heart block. Right auricle (1) and left ventricle (2) enlarged

The size of the heart at *cardioscopy* varies so that it may be normal or may appear small, but in the presence of considerable lengthening of the P–R period or heart block, moderate enlargement takes place (Fig 265)

Periodic paralysis

Familial periodic paralysis is closely related to potassium metabolism During the attacks the serum potassium value is halved, and characteristic changes in the electrocardiogram include prolongation of the P–R, QRS, and Q–T intervals, changes in the R–T segment, and diminished amplitude of the T waves. The changes are partially abolished by atrophine, and completely disappear at the end of the paralytic attack, whether spontaneous or brought about by the administration of potassium salts.

Diphtheria

The heart is commonly affected in diphtheria and it is always involved in fatal cases with the exception of the rare instances which succumb late in the illness to diaphragmatic paralysis When recovery from diphtheria does take place, however severe the illness has been, there remains no evidence of permanent heart damage.

The main pathological change consists of severe degeneration of the muscle, even to the stage of necrosis, while inflammatory and reparative processes are slight.

Clinically a triple rhythm due to addition of the third heart sound, or of the fourth heart sound if the P–R period of the cardiogram is prolonged, is a fairly constant sign, and tachycardia in the early stage of the illness often gives way to a slower heart rate in the later stage, and this should not be regarded as a favourable sign. Although the heart is commonly involved during the first week, as proved cardiographically, the signs of heart failure are usually absent, but in those patients who die in the second week or later, they are prominent.

On *cardioscopy* the cardiac silhouette is enlarged and in part this is due to the presence of pericardial effusion.

The *electrocardiogram* is often altered in diphtheria, and when the illness is judged to be severe on clinical grounds the tracing is nearly always abnormal. The changes include partial or complete heart block, bundle branch block, depression of the S–T segment, inversion of the T waves, auricular fibrillation and auricular tachycardia. This cardiographic test is so indispensable in diphtheria that hospitals, the special care of which is to treat infectious fevers, should always include the electrocardiograph amongst their equipment.

Anaemia

Palpitation, dyspnoea, and oedema of the ankles, are common symptoms in anaemia, and are more often the direct outcome of the blood condition than an indication that the heart is affected

Pain in the chest is another common symptom in severe anaemia, and this closely simulates cardiac ischaemia from coronary disease, but it differs from it in that it is of small import, it disappears with treatment of the anaemia, and it is not liable to the sequel of cardiac infarction. The knowledge that this symptom is not rare

in anaemia gains in value when it occurs in a female patient whose use of cosmetics has obscured some of the superficial signs of anaemia. Less often, ischaemia of the limbs may give rise to the symptom of intermittent claudication, but here, too, its significance does not bear comparison with the syndrome which results from atherosclerosis of the femoral arteries and their branches. When this symptom results from anaemia, pulsation continues to be present in the dorsalis pedis arteries.

The pulse may be collapsing in character and the pulse pressure increased from a lowering of the diastolic blood pressure. Haemic murmurs are common; a systolic murmur in the pulmonary area is louder than in the mitral area as a rule, and both are less well heard in the upright position than in the reclining posture.

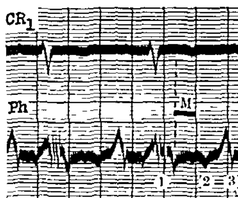


FIG 266—The haemic murmur (M) is seen to start in mid-systole

The haemic murmur is situated in mid-systole (Fig. 266). Another auscultatory sign found in anaemia is triple rhythm from addition of the third heart sound, and when this is present a measure of heart enlargement can be predicted as well as certain electrocardiographic changes Indeed, this triad of objective symptoms is the expression of right heart failure, with anaemia as its cause in this instance.

The *electrocardiogram* is often normal in anaemia, although in long-standing and severe cases, changes due to right heart preponderance may be present; these include inversion of the T wave in leads III and CR_1, and inversion of the T or depression of the R–T segment in lead II The S wave in lead I is seldom deep The correction of such irregularities does not always take place when the anaemia is successfully treated.

On *cardioscopy* the heart may show slight or obvious enlargement, both to the right and left In some cases it returns to normal size with the disappearance of the anaemia (Figs 267 and 268), but occasionally the enlargement remains

Vitamin deficiency

Although vitamin C can induce diuresis in a patient with fluid retention from heart failure its deficiency does not cause any changes in the cardiovascular system; the want of vitamin B can, however, produce such effects.

Amongst races accustomed to a primitive diet, deficiency of vitamin B_1 has sometimes been prevalent, giving rise to the symptoms identified with beri-beri. Apart from these circumstances, and in enforced or accidental famine, this vitamin want is seldom great enough to initiate symptoms, except that in patients addicted to alcohol the dietary may be so inadequate as to produce characteristic symptoms.

Symptoms.—These fall into two groups, the one resulting from alcoholism, and the other being the direct outcome of vitamin deficiency. Anorexia, nausea,



FIG 267.—Generalized enlargement of the heart from anaemia in a female aged 40 years.

vomiting, and epigastric pain, are symptoms identified with the continuous intake of alcohol Shortness of breath, distension of veins in the neck, enlargement of the liver, ascites, and oedema of the ankles appear as the result of right heart failure Anaemia and polyneuritis are sometimes present. The pulse is usually rapid and the blood pressure is low.

Cardioscopy shows moderate enlargement of the heart, chiefly of the right heart, and pulmonary congestion.

The *electrocardiogram*, although not distinctive, may show some or all of the following changes: low voltage, R–T deviation, and flattening or inversion of the T wave.

Treatment.—This consists of withholding alcohol and restoring a normal dietary At the start 2 mg. of vitamin B_1 should be given daily, intramuscularly, and mercurial diuretics twice weekly. Ascorbic acid (vitamin C) also proves a useful

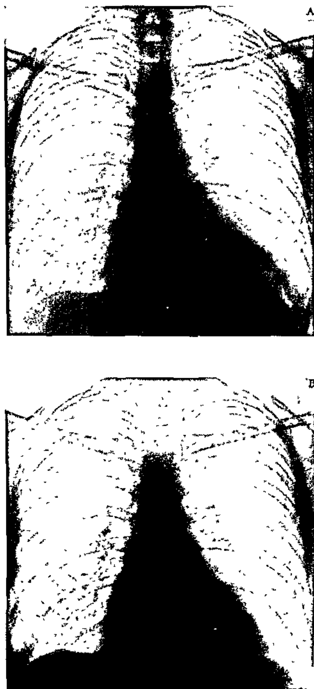


FIG 268—Enlargement of the heart (A) from anaemia in a female aged 41 years was much reduced (B) after two months' iron therapy.

diuretic during the first week, and iron should be added if anaemia is present When the response to treatment is satisfactory, the anasarca disappears and the enlarged cardiac silhouette seen on cardioscopy returns to normal , the results are often disappointing and the symptoms may progress in spite of the specific therapy.

Neurosis

To discuss neurosis in relation to the heart is to deal fully with what has become known as *Effort Syndrome*.

The diverse terminology applied to this symptom-complex is an index of its uncertain pathogenesis. Soldier's heart, athlete's heart, Da Costa's syndrome, and neuro-circulatory asthenia, are its other names. The last possesses the merit of directing attention away from the heart, because whatever hypothesis is brought forward to explain the nature of the disorder, one fact remains apart, namely, that the heart is healthy.

Symptoms and diagnosis—Giddiness or faintness, pain in the left chest, and palpitation, are characteristic symptoms Shortness of breath is not always among the presenting symptoms, but if it is made the subject of inquiry in one anxious to assent to unfitness, it is readily added to the other symptoms These complaints constitute an expression of nervous instability and are not the outcome of any heart affection. They are found in either sex, are more common in young subjects, and especially in those with poor physique who are emotional and hypersensitive, and sometimes choosing to lead a fallow existence The pulse is usually a little rapid from nervousness, and for the same reason the blood pressure is often slightly raised. The exercise tolerance test usually shows a poor response.

The *Exercise Tolerance Test* consists of observing the effect on the pulse and on the breathing of lifting the subject's own body-weight through a given height a fixed number of times at a standard rate. It is carried out as follows.

1 The pulse is counted for 15 seconds with the subject standing

2 The subject places one foot on a chair at least 15 inches in height, the other foot remaining on the floor He then raises himself so that both feet are on the chair, and then lowers himself so that both feet are on the floor again This movement is repeated 20 times in one minute

3. The pulse rate is then counted for 15 seconds

4. Immediately this has been noted, the breathing is watched to judge how far the exertion has produced distress.

5 After the subject has stood still for one minute after completion of the exercise, the pulse rate is counted for 15 seconds, by which time it should have returned to near the original rate

The data thus obtained are the responses of the heart and respiratory rates to exercise and rest The results should be recorded thus

E T T : 72-100-70, no D The figures indicate the pulse rate per minute on standing, immediately after exercise, and one minute after exercise, and no D (or D), the absence (or presence) of undue respiratory distress

The exercise tolerance test is of little or no value in the diagnosis of organic heart disease On the one hand, a patient with heart disease often performs the test satisfactorily, and on the other hand, a healthy subject will frequently show a poor response to the test

There is not any evidence of heart disease or of heart enlargement on clinical and radiological examination ; nor is there any evidence of thyroid toxaemia.

The electrocardiogram is physiological. Although the symptoms peculiar to the condition appear at odd times, they are voiced most ardently during periods of stress. To review them during selective phases in the life of a patient is to make the condition better understood, and to help allocate to it its true significance.

At school the subject's performance at games has been indifferent as a rule, and partly on this account and partly because of his physique the opinion of the School Medical Officer is often sought concerning fitness for games. Exemption from games has sometimes turned the subject more closely to his studies, but more often he has chosen to relax and obey his natural standards of ease and inactivity. Judicious management and encouragement by an understanding tutor at this stage has sometimes replaced his lack of self-confidence and vigour by a resolve to discharge creditably any responsibilities entrusted to him. More often, however, he has been left unnoticed to follow his own inclinations.

In civilian occupation the subject continues to lack confidence and stamina whenever the opportunity is offered to display them. On occasion, and particularly when a test of will-power is imminent, or during convalescence from an illness, he seeks treatment from his medical attendant. Reassurance is given, and a sedative medicine may be prescribed, or the heart may come under suspicion because of the presenting symptoms conjoined with the finding of a systolic murmur, and the foundation of cardiac neurosis is thereby unwittingly laid.

A subject presenting for medical examination in connexion with a Life Insurance policy remains silent about his symptoms. His pulse is considered rapid from nervousness, and as no evidence of organic disease is discovered on routine physical examination he may be classified a "first-class life," but if the tachycardia is associated with a trivial systolic murmur, and often *it is, the heart comes under unnecessary suspicion*.

It is during the medical examination preliminary to, or in the course of, military training that this symptom-complex looms prominently. The symptoms on this occasion receive emphasis by the recruit or soldier, and they claim unusual attention from the examiner, who finds that routine examination of young adults assembles a group presenting a common symptomatology. The examiner has to assign to each one a category as regards fitness for active military training. It is unwise to assess them as grade I in the first place because they will seldom grow into staunch combatants in that they are temperamentally unsuited to the calling and from the start they are opposed to a militaristic career.

From what has been said it is clear that this symptom-complex is in the nature of a neurosis and is not in any way to be regarded as a separate disease. Above all, it should not be thought to be some affection of the heart. The affliction is met with commonly amongst the civilian population, but when patients subject to it are convened in groups by military Medical Boards, it has often commanded greater attention than it merits.

Treatment.—The aim of treatment should always be directed towards creating within the individual that measure of will-power which will give him fuller mastery over himself and over his difficulties. It is often hard or even impossible to attain this end, and when circumstances have been such as to precipitate the condition it may be necessary to withdraw these even if it means that weakness appears to be thereby rewarded. Reassurance on the absence of heart disease must be given at the start. Encouragement should be given continuously, and sedative mixtures afforded a trial. Occupational therapy, embracing graduated exercises, has sometimes caused improvement.

CHAPTER 16

OTHER VASCULAR DISEASES AND EFFECTS

ISCHAEMIA OF THE EXTREMITIES

ISCHAEMIA of one of the lower limbs from peripheral vascular disease produces the familiar clinical syndrome known as *intermittent claudication* (intermittent limp) or angina cruris. Severe atheroma, associated with Monckeberg's calcification of the media or, less frequently, medial fibrosis of the arteries of the lower limbs including the femoral artery, supplies the common cause. The condition may be termed atherosclerosis The syndrome is also caused by thrombo-angiitis obliterans (Buerger's disease), but only rarely by Raynaud's disease for this involves the terminal parts of the arteries and the changes preponderate in the upper limbs

It used to be held that the pain was caused by anoxaemia of the muscles and nerves of a limb, but Lewis showed that the muscles elaborate a pain-producing factor which passes out into the tissue spaces and is normally removed by the blood-stream The development of pain is dependent on the accumulation of this substance, for if the circulation is inadequate, pain appears when the concentration of the pain factor has become excessive, and disappears only after a period of rest long enough to enable the blood-stream to remove the pain factor and to lower its concentration below the pain threshold It also plays a part in some cases of severe anaemia.

Aetiology

Ischaemia of the extremities from severe *atherosclerosis* affects males much more often than it affects females It is commoner in old age. In a recent analysis the age ranged from 41 to 76 years, 92 per cent of the subjects being over 50, 45 per cent in the sixties and 20 per cent in the seventies. It is a frequent complication of the chronic diabetes of old people, but in most patients there is not any diabetes and the cause is obscure.

In *thrombo-angiitis obliterans*, predominance of male patients is greater (90 per cent). Further, the subjects are younger, being as a rule between 25 and 50 years old. The earlier cases described were predominantly Jews, but this apparent racial predisposition is now considered to have been largely fortuitous In some cases the primary cause appears to be abrupt, probably allergic, degeneration of arteries The condition has long been attributed to tobacco smoking, but without convincing evidence Syphilis plays no part in either atherosclerosis or thrombo-angiitis obliterans

Raynaud's disease is much commoner in young women than in men. The discoloration of the limbs is readily induced by exposure to cold and consequently occurs more frequently in winter than in summer. Lewis held that impairment of blood supply to the digits in this condition was the result of closure of diseased digital arteries by intimal thickening He pointed out that the digital arteries possess this liability to closure in their whole length and that this can be annulled by the application of warmth He supported his view that the condition is caused by

local disease, and not by an abnormal response to the vasomotor mechanism, by the following experiment, if the vasomotor tone is deliberately reduced by warming the body of a patient with Raynaud's disease, immersion of the hand in cold water will still induce an attack. Again, if the hand is kept warm an increase in the vasomotor nervous tone brought about by cooling the body will not provoke an attack. Lewis held that the benefit resulting from ganglionectomy or preganglionectomy is due to the removal of normal rather than abnormal vasomotor tone, and that deliberate exposure of the limb to cold will still induce an attack in spite of the fact that the operation has brought about great clinical improvement.

Symptoms

In both atherosclerosis and thrombo-angiitis obliterans the lower limbs are more often affected than the upper limbs, but associated ischaemia of the upper limbs is much commoner in thrombo-angiitis obliterans than in atherosclerosis. Discomfort and fatigue in the muscles of the calf, behind the knee, and in the ankle or arch of the foot, associated with a sensation of cold, numbness or tingling in the feet, comprise the earliest symptoms. They are gradual in their onset and are provoked by exercise The condition progresses to typical intermittent claudication, when the patient, after walking a variable distance is forced to stop by severe cramp-like pain in the calf of the leg After resting for a few minutes the pain disappears, but recurs on walking In the later stages of the condition, pain may be present while the limb is at rest. In thrombo-angiitis obliterans the superficial veins may show thrombosis, giving a further source of pain associated with tenderness in the limb. A pink, red or even blue discoloration of the affected extremity is present, particularly when it is placed in a dependent position, and excessive pallor results from its elevation. Pain is rarely severe when the limb is at rest in atherosclerosis unless gangrene is present, but in thrombo-angiitis obliterans it may necessitate the limb being held in a dependent position, and this manoeuvre usually leads to oedema. Oedema is also caused in thrombo-angiitis obliterans by thrombosis of deeply seated veins. Trophic disturbances may develop so that callosities become prominent, and traumatic ulcers, if they occur, heal with difficulty. Ultimately, gangrene of one or more toes appears and may extend upwards Pulsation in the arteries to the distal portion of the limb, such as the dorsalis pedis artery, diminishes and disappears Oscillations determined by Pachon's oscillometer are smaller in amplitude in the affected limb. The skin temperature of the affected part is lowered ; this may be determined by the observer's hand, but lesser variations are best sought with a thermocouple type of skin thermometer. Atherosclerosis and thrombo-angiitis obliterans affect both legs, but one is usually more involved than the other. The more severely affected leg halts the patient when walking, so that subjective symptoms may not occur in the other leg. It is important, therefore, to estimate the condition of the circulation in both legs. An ergometer can be used to estimate the amount of work necessary to induce pain in either limb. Similar signs appear in the arms when they are affected.

The discoloration and coldness of the limbs in Raynaud's disease affects the hands to a greater extent than the feet, and the extremities of the digits are first involved before their proximal parts In the initial stages of the attack the digits become pale and then white and this phase is accompanied by a sensation of extreme

cold, numbness and pain. Later, the affected part becomes red and commonly shows cyanosis. Excessive sweating may be a troublesome complaint Atrophy may supervene, causing tapering of the fingers, and the skin becomes smooth and shiny. Necrosis of the nails or tips of the fingers is sometimes present, but frank gangrene is uncommon ; rarefaction of the bones may take place. Sometimes these phenomena are accompanied by scleroderma of the upper limbs or of the feet and face. Pigmentation may involve the affected area and arthritis may appear in several joints.

Differential diagnosis

In ischaemia due to *atherosclerosis* the patients are usually older, so that intermittent claudication in young male adults generally means *thrombo-angiitis obliterans*. Further, in atherosclerosis the pain is rarely severe during rest, and progressive thrombosis of veins does not occur. The commonest form of medial degeneration is Monckeberg's calcification, and if this is present it can be demonstrated by radiography. Medial calcification is not found in thrombo-angiitis obliterans.

Raynaud's disease usually attacks females, and the age incidence is lower even than in thrombo-angiitis obliterans Intermittent claudication is very rare. The colour of the limb is not markedly influenced by posture, but it changes after exposure to cold The upper extremities are more commonly affected, and gangrene is uncommon although necrosis of the terminal part of the fingers may sometimes occur. Pulsation in the arteries is not diminished and oedema and phlebitis are absent

Erythromelalgia is most commonly found in females The red and flushed limbs are not influenced by posture, pulsation is prominent, the temperature of the skin is high, intermittent claudication is absent, and gangrene does not occur.

These points in differential diagnosis are conveniently assembled in Table XV.

PROGNOSIS

The course of ischaemia of limbs varies If it is due to atherosclerosis the progress is usually slow and angina cruris may be present for many years before gangrene sets in. Some cases of thrombo-angiitis obliterans deteriorate rapidly, but others may show little deterioration over some years The outlook is best in Raynaud's disease. The eventual progress in any patient is judged by the extent of the condition and its response to treatment. Ischaemia of the arms as a result of thrombo-angiitis obliterans carries a grave prognosis, because in such cases the legs also are extensively involved.

TREATMENT

Particular care should be taken of the affected extremities. They should be washed and dried thoroughly twice a day and rubbed gently with hydrous lanolin The nails need careful attention, but minor surgical measures upon nails and callosities are to be deprecated because of the danger of precipitating gangrene. In cases of Raynaud's disease woollen hose should be worn, shoes should fit properly, and the hands should be clad in warm woollen gloves in cold weather. Warm

underclothing is also necessary Whenever possible a period of rest proves beneficial to the lower limbs when involved by atherosclerosis or thrombo-angiitis obliterans

TABLE XV

Outlining the main features in ischaemia of limbs due to atherosclerosis, thrombo-angiitis obliterans and Raynaud's disease.

Clinical features	*Atherosclerosis*	*Thrombo-angiitis obliterans*	*Raynaud's disease*
Sex	Males (90 per cent)	Males (90 per cent)	Females (90 per cent)
Age	55 or over	25 to 45	17 to 35
Claudication	Common	Common	Absent
Pain at rest	Slight	Severe	Usually absent
Arterial pulsation	Diminished or absent	Diminished or absent	Normal
Excessive rubor of limb with dependency	Present	Present	Absent
Excessive pallor of limb with elevation	Present	Present	Absent
Colour changes on exposure to cold	Sometimes	Sometimes	Always
Temperature of extremity	Low	Low	Low
Temperature of extremity after induced fever or sympathetic block	Rarely raised	Often raised	Always raised
Incidence of gangrene	Common	Common	Rare
Kind of gangrene	Dry	Moist with surrounding inflammation	Punched-out areas
Superficial phlebitis	Absent	Present in one-third	Absent
Oedema	Infrequent	Frequent	Absent

Medicinal remedies have been disappointing in the treatment of ischaemia of the limbs. Vasodilator drugs, such as glyceryl trinitrate and acetylcholine, have not proved valuable, and extracts of muscle and pancreas are without benefit. Thyroid may occasionally give temporary benefit in patients with Raynaud's disease, but it is never curative.

Measures should be adopted which may increase the efficiency of the collateral circulation to the diseased limb. Such measures are of undoubted value in thrombo-angiitis obliterans, because in this condition the arteries which do not show thrombosis may increase in size to form a collateral circulation, as is seen in arteriograms. Atherosclerosis, however, is so wide in its distribution that there can be little hope of improving materially the collateral circulation. The

media of the arteries shows degeneration, so that methods to induce vasodilatation, notably lumbar sympathectomy, cannot often prove valuable Postural exercises consisting of alternatively elevating and lowering the limb may produce benefit when carried out at regular intervals each day. Heat applied to the limb through the medium of electric pads, or other means, often proves harmful, and the direct stimulus of cold is more likely to give benefit. Good results can sometimes be obtained by providing warmth to the body so as to encourage vasodilatation in the limb This is also sometimes brought about by immersing the limb alternatively in hot and cold water baths Non-specific protein therapy, beginning with a dose of 20 million T.A B, given intravenously, has sometimes proved beneficial. Frequent intravenous injections of hypertonic saline have been advocated, and more lasting benefit is expected from transfusion with blood serum, which promotes collateral circulation by increasing the blood volume; some 800 cc. may be given each time over a period of up to two hours. The diseased limb can be introduced through a carefully made cuff into a strong glass casing from which air is removed and introduced to cause alternating negative and positive pressures of 25 seconds' duration (Pavex apparatus) so that the blood is drawn into the tissues from the arteries and then expelled through the veins Intermittent venous occlusion by cuffs inflated and deflated by a simple syphoning device connected with a water tap often produces clinical improvement in patients with intermittent claudication from atherosclerosis and sometimes thrombo-angitis obliterans.

If gangrene develops, amputation may become necessary, but conservative methods often result in separation of the infarcted area with healing. The lowest level for amputation is best determined by applying the reactive hyperaemia test.

In Raynaud's disease, and especially in those cases of ischaemia of the limbs from thrombo-angiitis obliterans where a vasoconstrictor effect has been demonstrated, the operation of sympathectomy or ganglionectomy has sometimes produced a lasting benefit. This procedure is not expected to benefit cases of ischaemia of the limbs due to atherosclerosis.

In lumbar ganglionectomy the removal of the second and third sympathetic ganglia interrupts the pre-ganglionic fibres to the sciatic nerve and produces favourable results in the selected cases. Cervico-thoracic ganglionectomy, on the other hand, has produced only temporary benefit when employed in patients presenting Raynaud's syndrome, and this has been explained by the fact that cervical ganglionectomy only interrupts the post-ganglionic fibres, better results are anticipated from cervical pre-ganglionectomy, which also obviates the production of disfigurement associated with Horner's syndrome. This is brought about by section of the sympathetic cord below the third thoracic ganglion, together with division of the rami of the second and third thoracic nerves, but this form of operation is still under trial, and the results so far are not uniformly good.

Two methods may be used to test the immediate efficiency of the operation The first depends on the absence of sweating over the area supplied by the divided sympathetic fibres, and this is tested by inducing sweating and noting a change in colour in strips of cobalt-blue paper distributed over and near the area to be tested The second method depends on the observation that as sympathetic

nerve fibres to a limb degenerate, the denervated smooth muscle of the arterial walls become hypersensitive to adrenaline. Thus, when a solution of 1 part of adrenaline in 250,000 parts of normal saline is given intravenously at the rate of one drop a second, it will cause vasoconstriction with a fall of temperature in a denervated limb after a successful operation.

HOW TO TEST THE EFFICIENCY OF THE BLOOD SUPPLY TO A LIMB

The methods which may be employed to estimate the efficiency of the circulation in a limb are many. They concern the rate of the blood flow, the state of the vessels and their capacity to dilate, or the efficiency of the vasoconstricting and vasodilating apparatus.

Colour of the skin.—Discoloration of the skin provides valuable information concerning the condition of the blood supply to the part The colour of the limbs is first noted when the patient is in a reclining position with the extremities lying level with the body. The colour changes are then observed when the limb is elevated and again when it is lowered to a dependent position With a deficient circulation it assumes a deep pink or bluish-red colour in the dependent position and excessive pallor or blanching when it is held in the elevated position

Allen's test.—In the case of the hand, Allen described a simple method of detecting obstruction in the radial or ulnar artery at or distal to the wrist After the patient has clenched both fists firmly for about half a minute, both radial pulses are obliterated at the wrists by digital compression The patient then relaxes his grip When disease has caused obstruction in the ulnar artery the colour returns slowly to the hand until the radial artery is released Similarly the blood flow through the radial artery is tested following compression of the ulnar artery. Although the rate at which the colour returns to the hands in health varies, it should return quickly, and the value of the test lies in finding a difference in the rate in the two hands

Reactive hyperaemia.—This test combines the advantage of simplicity and efficiency The limb to be tested is first immersed for ten minutes in hot water to ensure relaxation of any vascular spasm that may be present It is then raised above the level of the body and massaged until the smaller vessels become emptied of blood and the skin is pale The circulation is then arrested for ten minutes by inflating a pneumatic cuff around the upper end of the limb to a pressure beyond the systolic blood pressure, while the limb is again immersed in the warm bath This procedure brings about efficient dilatation of the vessels by depriving the tissues of nutrition The limb is then removed from the bath and the cuff is deflated. If the arteries are healthy the skin becomes brightly coloured as far as the ends of the digits within five seconds

Arterial pulsation—Obstruction within an artery reduces or obliterates the pulse distal to the seat of obstruction The pulse disappears before the blood flow ceases and the flow may continue long after the obstruction has taken place because a collateral circulation supplies the artery distal to the seat of obstruction In the upper limb the pulse is sought in the axillary artery, in the brachial artery along its whole course, and in the radial and ulnar arteries at the wrist In the case of the lower limbs pulsation should be recorded in the femoral artery, in the posterior tibial artery behind the internal malleolus and in the dorsalis pedis artery in a line passing between the bases of the first and second metatarsals A pulse should not be regarded as absent until examination has failed to detect it in a warmed limb Arterial pulsation may be recorded by Pachon's oscillometer or a Tycos manometer, and the readings obtained should be calibrated against similar readings from the opposite limb and from a healthy subject

Condition of the arterial wall.—Disease of the arterial wall can often be detected by palpation. If medial calcification is present, the artery feels like a pipe-stem With any medial degeneration the artery tends to be irregular, and tortuous In pure medial hypertrophy the arteries feel abnormally large, firm and elastic The firmness varies from time to time, being greater the more the artery is contracted Estimation of the blood pressure will decide whether or not hypertension is a factor in producing arterial changes Calcareous deposits in arteries may be demonstrated in teleradiograms, and the femoral and posterior tibial arteries should always be subjected to x-ray examination whenever efficiency of the circulation to the lower extremity is to be estimated.

Observation on the surface temperature of the limbs.—Variations in temperature are greatest in the most distal parts of the extremities and are less as the proximal part of the limb is

reached, so that the temperature of the digits provides the most sensitive record of changed temperature within a limb resulting from alteration in the circulation. The recorded temperature of a limb suspected of an impaired circulation should be compared with that obtained from the opposite limb and from the corresponding limb of a healthy subject. Although the observer's hand when warm can appreciate small differences in temperature between different areas of a limb, a skin thermometer is necessary to record accurate values.

The rate of heat elimination in a limb can be estimated by a calorimeter. Thus, the foot is immersed in a known volume of water at a temperature slightly lower than that of the skin and the temperature of the water is recorded at regular intervals and the results plotted.

The value of recording skin temperature is greatest when estimating the capacity of the blood vessels in a limb to dilate and the efficiency of the vasoconstrictor and vasodilator mechanism. The following methods of removing vasomotor tone to the peripheral blood vessels have been applied and although each is efficacious some are cumbersome to carry out and involve discomfort to the patient. In all the tests attention should be paid to the height to which the temperature of the limb rises as well as the rate at which the rise takes place.

In the upper limb the vasoconstrictor fibres may be blocked by anaesthetizing either the nerve trunk or the stellate ganglion, but obviously this is not a convenient method.

The vasoconstrictor fibres to the lower limbs can be blocked by inducing spinal anaesthesia so that complete vasodilatation is brought about in the legs. If obstruction to the circulation has been due to angio-spasm the latter is immediately removed so that the temperature of the legs rises abruptly and noticeably. If, on the other hand, the obstruction is caused by organic changes in the artery and there is severe degeneration of the media, there may be little or no vasodilatation.

Vasomotor tonicity in the skin vessels of a limb may also be abolished by heating the body and this may be brought about in diverse ways. In all the methods used, the warmed blood reaches the heat-regulating centre which is sensitive to changes in temperature. A lowering of the vasomotor tone results and the skin becomes flushed and the surface temperature rises. If the blood supply to the skin is impaired by obstructive vascular disease the rise in skin temperature is not appreciable and is delayed. Some four different ways of producing vasodilatation in the limbs through heating the body have been tried.

While the limbs on which surface temperature observations are to be made are exposed, the trunk is enclosed in a chamber heated with lamps to a temperature of about 60° C.

Again, the limbs on which observations are not being made are immersed in water at a temperature of 44° C. while the skin temperature is recorded in the diseased limb or limbs.

Systemic pyrexia can be induced by intravenous injections of T.A.B., consisting of about 20 million bacilli, and surface temperature observed in the diseased limb or limbs. Alternatively intravenous injection of Pyrifer may be given.

With the diseased limb or limbs exposed in a room at a constant temperature, the systemic temperature may be raised by covering the trunk with blankets kept warm by hot water bottles. This is a convenient and effective method.

From any of these tests it is possible to estimate the vasomotor index for each limb. In subjects with thrombo-angiitis obliterans, unless a good vasomotor response is recorded, lumbar ganglionectomy is not likely to prove of permanent benefit.

When intermittent claudication is present as a symptom of arterial disease in the lower extremities, the extent of the circulatory impairment can be estimated by testing the movement of the foot with an *ergometer*. Thus, the leg is rested on a back splint and is slightly elevated in order to ensure adequate venous return and the thigh is anchored down so as to prevent flexion of the knee and use of the thigh muscles. The foot is strapped to a hinged foot-piece and is made to raise a weight of 5 pounds during plantar-flexion; the patient is requested to extend and flex the foot at a rate governed by a metronome. The number of times the weight is elevated before discomfort is felt in the calf is counted and this is compared with results obtained in the symmetrical limb. The test may be applied periodically to estimate the benefit gained from any form of treatment which has been adopted.

Arteriography.—By radiographing a limb at a suitable interval after injecting into a large artery a substance opaque to x-rays, the size of the arterial lumen, points of obstruction, and the state of the collateral circulation can be determined. strontium, iodine, and thorium compounds have been used for this purpose. The method is not without risks.

ARTERIO-VENOUS ANEURYSM

When a communication is established between an artery and a vein the condition is referred to as an arterio-venous aneurysm Its differentiation into aneurysmal varix, in which the two vessels anastomose directly, and varicose aneurysm, in which the connecting vessels are separated by a sac, is of doubtful value, because each type has a common aetiology and it is only possible to define the particular variety after examination of the resected specimen. Arterio-venous aneurysms usually result from trauma, but sometimes they follow inflammation. A congenital variety is also met with in which multiple arterio-venous connexions give it the appearance of a diffuse angioma A communication between the cavernous sinus and the internal carotid artery as it passes through the sinus may follow a fracture of the base of the skull when the fistula is established either immediately the fracture has occurred or some days or weeks later. Arterio-venous aneurysm has sometimes followed clumsy venesection at the elbow. When situated in the thigh it is often the result of shrapnel or bullet wounds. Occasionally it has been found in the foot and has then followed injury, or less commonly gangrene Rarely, a communication has taken place between the ascending aorta and the superior vena cava or pulmonary artery, in aortitis.

SYMPTOMS AND DIAGNOSIS

Headache, depression and lassitude are present as general symptoms in arterio-venous aneurysm, but its precise symptomatology varies greatly and depends on the site of the lesion and the size of the fistula. Thus, when situated in the cavernous sinus it produces exophthalmos and the protruding eye pulsates ; if the pulsation is not visible it can be elicited by pressing lightly on the globe of the eye. Owing to its position within the cranium the loud murmur associated with the aneurysm causes great distress to the patient. When the aneurysm is situated in a limb, oedema of the distal portion and even ulceration of the skin may result. If a communication exists between the aorta and superior vena cava, cyanosis and oedema of the face are presenting features Whenever the lesion is accessible to the examining finger a pulsating swelling can be made out and the artery above is dilated showing increased pulsation The oscillometer will register increased excursions. A systolic thrill is felt both over the aneurysm and over the artery for some distance proximal to the lesion. A loud and rough systolic murmur has a similar distribution If the fistulous opening is large, the pulse pressure is increased and the pulse is collapsing in character. Obliteration of the arterio-venous shunt by digital compression raises the systolic blood pressure (temporarily) and the diastolic blood pressure to a greater extent, and the pulse pressure is thereby lessened, abolishing the collapsing nature of the pulse An appreciable fall in the pulse rate also takes place when the arterio-venous fistula is obliterated, because of a decrease in the venous return to the heart. As long as the fistula remains open the general venous blood pressure is considerably raised Dyspnoea, palpitation, cough and evidence of heart failure appear when the fistulous opening is large and the condition established for some time. The apex beat is forcible and displaced outwards. A systolic murmur may be heard over the heart. Cardioscopy will demonstrate generalized enlargement of the heart and congestion of the hilar vessels These changes disappear within a short period of the closure of the fistula (Fig. 269). Whenever a patient is breathless and shows cardiac enlargement



Fig 269 —Arterio-venous aneurysm Enlargement of the cardiac silhouette involving the right auricle (1), and the left ventricle (2), and hilar congestion (3), shown in (A) disappeared a month later (B after removal of a femoral arterio-venous aneurysm

and a collapsing pulse in the absence of an aortic diastolic murmur, an arterio-venous aneurysm should be sought. Sometimes a teleradiogram of the part carrying the fistula has shown calcification within the aneurysm, and an arteriogram can demonstrate the actual fistulous opening.

TREATMENT

In the intracranial variety, radical cure by extirpation of the aneurysm is not possible, but ligature of the internal carotid artery on the side of the lesion may diminish the loud systolic murmur which is so distressing to the patient; the procedure may, however, sometimes precipitate hemiplegic disturbances If the site of an arterio-venous aneurysm permits easy access, operative extirpation or closure of the communication cures the condition The symptoms of heart failure are relieved, the size of the heart is diminished, the systolic blood pressure falls to normal after the initial rise, the diastolic blood pressure is raised to its normal level, oedema in the distal portion of the affected limb disappears, and ulcers heal rapidly Improvement in the general health and well-being of the patient is a noticeable post-operative feature

RUPTURE OF THE AORTA

Rupture of the aorta producing the syndrome known as *dissecting aneurysm* is seen in two forms In the one, severe medial degeneration is associated with considerable or severe atheroma . the patients are usually males, and cardiovascular hypertrophy is almost always present In the other, there is very little or no atheroma, the aorta in the neighbourhood of the rupture is smooth, thin, and dilated, the patients are as often females as males, and there need not be hypertension In either case the blood entering at the rupture tears a passage within the media or separates the media from the adventitia or intima Different kinds of dissection may occur in different parts of the extension. The new channel usually enters the lumen again at one or more orifices of exit. The site of election of the rupture, especially in the second type, is the ascending aorta. If the dissection passes towards the heart, it usually ruptures within a short time through the adventitia into the pericardial sac, causing haemopericardium and death If the dissection passes caudalwards it may extend for a very great distance, for instance, from the ascending aorta to reach the iliac and femoral arteries. Death is usually caused sooner or later by the dissected channel rupturing through the adventitia into the pleura, lung tissue or retroperitoneal tissue, or cutting off the circulation from branches of the aorta. Occasionally, however, the patient survives, the rupture is healed by scar tissue, the dissected channel remaining confined to a short extent of media, or a long dissected channel becomes a walled vessel, a " double aorta " being formed

The symptoms caused by the rupture are ushered in suddenly by intense pain in the chest and back which often compels the patient to scream with anguish The severe pain is associated with extreme shock, dyspnoea, and often with loss of consciousness followed quickly by death. Often the pain is situated in the upper abdomen. In most cases the course of the disease is so rapid and death follows so quickly that there is little opportunity for the elicitation of physical signs. Pallor is usually a feature and is the result of shock, but cyanosis of the face and neck has occasionally been noticed from obstruction of the superior vena cava when

the dissection has implicated the ascending aorta. Haemoptysis occurs if the aneurysm ruptures externally into the lung tissue, and on occasion haemorrhage from the bowel has followed infarction of the intestine caused by interference with circulation in the superior mesenteric artery. Vomiting is sometimes present Hypertension is usually present in the first type. Symptoms suggesting a cerebral catastrophe may appear on account of interference with the commencement of the carotid arteries. When the rupture has occurred externally into the pericardium the patient seldom survives long enough for evidence of haemopericardium to be made out. In a few cases the radial or femoral pulse has been absent on one or both sides. A murmur has been heard over the ascending aorta, caused by vibration of the edge of a transverse rupture. Cardioscopy before symptoms have arisen might demonstrate the aortic dilatation and elongation, as well as enlargement of the left ventricle.

DILATATION OF THE PULMONARY ARTERY

It is rare for increased pulsation in the left second intercostal space to be noticed in dilatation of the pulmonary artery, and cardioscopy affords the only certain means of establishing its presence. Enlargement of the pulmonary artery is recognized by finding the pulmonary arc conspicuous in the anterior and right oblique positions, the pulmonary ovoid (right pulmonary artery in section) dense in the right oblique position, and the pulmonary arch (left pulmonary artery) outlined below the aortic arch in the left oblique position. In many instances of pulmonary artery enlargement, the conus of the right ventricle is also prominent from hypertrophy of the right ventricle. The causes of pulmonary artery distension have been discussed separately in the context, but a summary of these might be useful.

1. Relative prominence of the pulmonary arc on cardioscopy is seen in young adults with a long thorax and vertical cardiac silhouette. It is also found in young healthy subjects in whom the thorax appears normal Again it is conspicuous in children in whom the cardiac shadow is squat, assuming a globular shape.

2 Dilatation of the pulmonary artery is found in about one-fifth the cases of congenital pulmonary stenosis ; hypoplasia is present in the remaining four-fifths Rarely, a defect of the ventricular septum, biventricular aorta and hypertrophy of the right ventricle are associated with congenital dilatation of the pulmonary artery (Eisenmenger's syndrome).

3 Moderate and sometimes considerable dilatation of the pulmonary artery occurs when a patent ductus arteriosus establishes an aortic-pulmonary arterial shunt.

4. The pulmonary artery is always larger than the aorta in cases with congenital defect of the auricular septum. The dilatation of the pulmonary artery and its branches which occurs in this condition is usually considerable and often extreme, and is associated with pulmonary incompetence

5. "Idiopathic dilatation" of the pulmonary artery due to a congenital defect of the arterial wall or to an unequal division of the truncus arteriosus has sometimes been described, but many of the reported cases have been instances of pulmonary artery dilatation from auricular septal defect when examined at necropsy.

6 Enlargement of the pulmonary artery is a common finding in mitral stenosis, and this change is associated with enlargement of the conus and body of the right ventricle.

7. Moderate enlargement of the pulmonary artery is found in a proportion of cases of thyroid toxaemia. When this is associated with generalized cardiac enlargement the appearance of a " ham-shaped " heart is presented.

8 Rarely, an aortic-pulmonary arterial shunt producing enlargement of the pulmonary artery is caused by rupture of an aortic aneurysm into the pulmonary artery

9. In emphysema associated with conspicuous cyanosis there may be selective enlargement of the pulmonary artery in the absence of much cardiac enlargement.

10. Primary pulmonary hypertension is another uncommon cause of considerable enlargement of the pulmonary artery, and associated in this instance with enlargement of the right auricle and right ventricle

TORTUOSITY OF ARTERIES

Tortuosity of arteries is a feature of atherosclerosis and of hypertension In hypertension the deformity may sometimes result directly from the raised blood pressure and is due to physiological propulsion, but atherosclerosis is commonly superadded and in this instance the tortuosity takes the form of a lasting arterial deformity. It is common to find tortuosity of the radial, brachial, and temporal arteries in older subjects, but this finding is without any great clinical significance. A kinking of the right carotid artery in the neck assumes importance in that it has to be considered in the differential diagnosis of aneurysm of the carotid artery. Hypertension is usually present in a female patient with a kinked carotid and the tortuosity is then produced in its greater part by elevation of the aortic arch within the thorax, a change which is part of the uncoiling of the thoracic aorta taking place in hypertension. In a few cases, kinking of the aorta is present in the absence of hypertension and the deformity then is the result of atherosclerosis. The condition does not count in prognosis. Cardioscopy in the three orthodox positions will sometimes show sharp kinking of the descending thoracic aorta affected by *atherosclerosis, and the hoop-like formation of the upper moiety of the thoracic* aorta in hypertension is a familiar finding. In the anterior view this shows as a wide vascular pedicle, raised aortic arch, and prominence of the descending aorta to the left which meets the heart shadow at a lower level than in the normal In the left oblique view the hoop effect is well seen, and the aortic window (in the region of the left bronchus below the aortic arch) is large, while the base of the aortic triangle above is lengthened In the right oblique position the aortic shadow is farther to the left than normal, but it does not show clubbing as in aortitis These changes assume importance in diagnosis and take no part in assessing prognosis. The appearance of the aorta in kyphosis resembles in some ways the aorta of hypertension and atherosclerosis. Its recognition comes from noticing the spinal deformity.

PERIARTERITIS NODOSA

The changes in the arteries in this condition are those associated with the process of acute degeneration and necrosis. The arterial changes may be found alone or may complicate some other disease, particularly nephritis Periarteritis nodosa is

uncommon, but its actual incidence is higher than is suggested by the infrequency with which it is diagnosed clinically. It is sometimes found unsuspectedly in tissue removed at operations. The focal necrosis of the small and medium arteries leads to aneurysmal dilatation, haemorrhages and infarction, particularly in the kidney Some cases may be described as fulminating, because coagulative necrosis of arteries has been severe and widespread, leading to early death. In such cases there may be gangrene of the extremities. More often the condition is less acute, and progression is shown by the presence of reparative processes such as organization of thrombi and fibrosis of infarcts. Many arteries are usually affected and any of medium or small size may be involved. The arteries most commonly affected are the renal, hepatic, pancreatic, coronary, splenic and mesenteric Involvement of arteries to spinal roots and nerves is not uncommon, causing obscure nervous disturbances. The brain is less commonly affected, but when arterioles within the cerebral substance are involved, the condition appears as multiple small haemorrhages. The arteries in the lung are seldom affected Involvement of the pancreatic arteries causes acute pancreatitis.

SYMPTOMS AND DIAGNOSIS

According to the extent and distribution of the arterial lesions the symptoms vary widely. The disease may be ushered in with irregular pyrexia, tachycardia, and extreme prostration. Dyspnoea, cough and haemoptysis are not infrequent, but although cardioscopy may reveal conspicuous pulmonary oedema, the finding of the specific changes in the pulmonary arteries is rare. Involvement of the mesenteric artery may cause severe abdominal pain, vomiting and diarrhoea or infarction of the intestine resulting in perforation and peritonitis. Involvement of the coronary artery may cause cardiac infarction. In some cases, the condition complicates nephritis, particularly the so called nephrosclerosis The changes of hypertensive retinitis may be present, but these differ in no way from similar changes associated with hypertension of other origin. Although a variable degree of hypertension is present in cases complicating nephritis it is not essential in periarteritis nodosa. Occlusion of the central artery of the retina has been described and histological sections have shown arterial changes in the choroid. In a small proportion of cases subcutaneous nodules of varying size up to a pea can be felt over the trunk and limbs causing generalized pain and discomfort, when these are present, biopsy provides a certain method of establishing the diagnosis A blood count shows diminution in the number of red cells and the haemoglobin value, as well as moderate leucocytosis, eosinophilia is sometimes present Blood cultures have so far proved to be sterile

Owing to the variable distribution of the lesions the symptoms are thus seen to be protean. Often the cause of an obscure illness characterized by pyrexial tachycardia, pulmonary symptoms, albuminuria and prostration, is only discovered to be periarteritis nodosa at necropsy. Sometimes a subcutaneous nodule is found during life and its histological examination establishes the true nature of the condition.

PROGNOSIS

In the majority of cases death takes place within a few weeks or months of the onset of symptoms. From findings at biopsies and necropsies, however, there is

evidence that the condition may progress slowly and that even spontaneous healing and recovery may take place in milder cases.

No form of specific treatment has proved of any value in this condition so that measures similar to those in general infections should be applied, and symptoms dealt with as they arise.

Temporal Arteritis

Temporal arteritis is a rare condition affecting patients in the fifth or sixth decade *Headache is the principal symptom, chiefly referred to the site of the affected vessels; it is* aggravated during movement of the face or jaw as in speech or mastication There is thickening of the vessels which gradually show loss of pulsation The disease lasts for several months and recovery is usual Diagnosis rests upon a biopsy and this procedure may relieve the symptoms as well

The histology of the condition is that of an arteritis, unrelated to tuberculosis or syphilis The intima is greatly thickened and thrombosis may take place within the reduced lumen The media is largely replaced by granulation tissue which may show characteristic giant cells with many nuclei

EMBOLISM

Embolism manifests itself suddenly When the accident happens it creates a state of emergency on account of the decision to be made on surgical treatment which has to be applied early to be successful, and on account of the difficulty in assessing prognosis Its seriousness is connected with its effects at the site of impaction, with the nature of the lesion providing the source, and to a lesser extent with the character of the embolus.

THE EMBOLUS

Usually the embolus comprises either the whole or part of a thrombus, situated in the venous system or within the heart cavities enlarged from valvular disease, hypertension or from a developing cardiac aneurysm Thrombosis within an aortic aneurysm provides another source of embolism, but it is a rare event as the result of atheromatous changes The embolus sometimes consists of septic material and examples of this are provided by brain abscess in bronchiectasis, visceral abscesses in pyaemia, and septic infarcts, mycotic aneurysms, and subcutaneous nodules in bacterial endocarditis Groups of neoplastic cells may also produce embolic effects

Fat embolus and embolism.—Injuries frequently include simple or compound fractures of bones, and widespread laceration of fat-laden tissues The condition is not always recognized clinically and even at necropsy it remains undetected unless sections of the affected part are specially prepared It has been shown that slight grade fat embolism occurs in about 10 per cent of deaths from causes other than injury, but severe or moderate fat embolism is the result of injury, and when demonstrated at necropsy it is evidence that the trauma happened during life In 20 per cent of all patients with fracture of the lower limbs who die within three weeks of the injury, severe fat embolism is present and is the direct cause of death in 5 per cent The source of the embolus in this instance is probably in the fat depot in the bone marrow When the fat reaches the venous system it is carried into the lungs, resulting in embolism and it interferes with the pulmonary circulation giving rise to dyspnoea, cyanosis and a fall in blood pressure In the majority of patients the emboli advance through the pulmonary capillaries into the systemic circulation In the systemic system the brain is the only vulnerable tissue, and cerebral infarction involves the white matter, resulting in areas of necrosis Although fat embolism of the heart has been described it is a rare event and only the pulmonary and cerebral varieties need to be considered.

The clinical picture produced by fat embolism is the outcome of both pulmonary and cerebral involvement Some three days after the injury, which usually includes a fracture of a lower limb, pyrexia and tachycardia set in, and petechial haemorrhages appear over the

chest Dyspnoea, cyanosis, cough, and crepitations over the lungs, tell of pulmonary involvement Restlessness, insomnia, delirium, stupor and coma, supply evidence of cerebral injury, but, as a rule, there are no localizing neurological signs Confirmation of the clinical diagnosis comes from radiological changes in the lungs in the form of a general haziness with scattered shadows, and from the finding of fat globules in the urine and more rarely in the sputum and blood

In the treatment of fat embolism, oxygen administration is helpful An intravenous drip infusion of sodium desoxycholate given two-hourly in a dose of 10 cc of the 20 per cent solution is under trial; otherwise the treatment is wholly symptomatic

Air embolus and embolism.—During manipulative or operative measures practised in the investigation or treatment of disease, the entry of air into the venous or arterial system is not unusual If the column of air is small and its introduction gradual, no symptoms follow and the air is absorbed by the blood in from ten to thirty minutes This explains the absence of untoward effects from the common procedures of intravenous therapy and venesection Should a large volume of air enter a vessel suddenly, severe and even fatal symptoms may arise Air embolism has been reported in operations on the neck, after Eustachian or Fallopian tube insufflation, and in pneumoperitoneum, but only two classes are discussed here, namely, manual separation of the placenta, and the induction of pneumothorax, because they provide examples of the two types of air embolism, the pulmonary, and cerebral type *In the less common pulmonary variety, air enters the pulmonary circulation by way of the* right heart, causing sudden pain in the chest and distressed breathing, death takes place *from failure of the right heart It is known that air bubbles in the pulmonary circulation* do not filter through into the systemic arterial system The frequency of operations on the lung and pleural cavity *accounts for the higher incidence of the cerebral variety* Although in the experimental animal the effects of systemic air embolism are seen in the coronary as well as the cerebral circulation, in man cardiac effects are not demonstrable Among the cerebral symptoms are pallor, headaches, nausea, vertigo, temporary blindness, convulsive tremors, and coma

When the catastrophe has taken place the patient's head should be lowered for at least half an hour after the symptoms have passed *Artificial respiration may be necessary* Adrenaline appears to have proved useful sometimes Treatment of air embolism should be directed chiefly to its prevention, and it is necessary to adopt certain precautions during the induction of pneumothorax A radiological survey is a preliminary need, for the pneumothorax needle should steer clear of areas of consolidation, cavitation, and adhesions, the danger of air embolism arises from a penetration of the visceral pleura and a piercing of the lung substance If retraction of the syringe plunger produces a frothy blood-tinged fluid, another puncture site should be selected Air should not be introduced until the needle aspirates air freely when the plunger is withdrawn and until the manometric oscillations are satisfactory

THE SOURCE OF THE EMBOLUS

Thrombus in the veins of the lower extremities is a frequent cause of embolism. *Indeed*, at necropsy, when other more accessible sites *have* failed to disclose the source of the embolus, it is customary to seek thrombosis in veins of the lower limbs, even though no clinical sign has suggested its presence in life. There is good authority *for the belief that although fulminating emboli often spring from the* femoral veins, thrombosis here represents an extension from older clots in the legs and feet. Venous thrombi tend to propagate towards the heart so that the most recent clot forms proximally. From the standpoint of prevention and treatment, recognition of this is important.

Although thrombosis within an aortic aneurysm or formed in connexion with an atheromatous ulcer in *the aorta has been known to give rise to embolism*, it is a rare event and heart disease is the only other common source of the embolus. Nearly one-half of all cases of heart disease show evidence of embolism at necropsy Bacterial endocarditis is the usual heart disease to associate itself with infarction of viscera, and 80 per cent of the cases show this complication About one-half *the cases of cardiac infarction at necropsy show embolism in the lungs or systemic* circulation In this instance intracardiac thrombosis takes place in the absence

of cardiac enlargement, and embolism is a more common event in early than in late cardiac infarction in which cardiac aneurysm has developed. Pulmonary and systemic embolism is not uncommon in prolonged hypertension, but less common than in mitral stenosis It is unlikely that embolism in mitral stenosis, in which it is pulmonary as often as systemic, occurs only when certain criteria are present, and there are many exceptions to any fixed views which might be held on the relationship between the two conditions. Nevertheless, clinical experience and the findings at necropsy permit certain general statements on the circumstances which appear to influence the formation of thrombosis and embolism in mitral stenosis. In certain instances of ventricular thrombosis there is evidence that embolism follows soon after the thrombosis, but no such information is available for auricular thrombosis The effect of auricular fibrillation on the formation and dispersal of a thrombus is also uncertain. The higher incidence of embolism in patients with fibrillation may be explained by the longer duration of the valvular lesion in those presenting an abnormal rhythm, and not by the actual arrhythmia Furthermore, embolism is common in cases which preserve a normal rhythm. In patients in whom embolism develops within a short time of a return of fibrillation to sinus rhythm during quinidine therapy, it is right to attribute the complication to the change of auricular action

The formation of a ball or mass thrombus in the left auricle in mitral stenosis sometimes produces a temporary major embolic effect when it comes to lie astride the narrowed mitral orifice ; this induces cardiac ischaemia precipitating pain in the chest, and less commonly ischaemic changes in the extremities as well.

THE SITE OF IMPACTION

Cerebral embolism—It is sometimes difficult to tell cerebral embolism from cerebral haemorrhage, but usually it is possible to recognize the nature of the injury when all the signs are considered In embolism the onset of the attack is more sudden than in arterial rupture, loss of consciousness may be transient, recovery of speech more rapid, and the resulting hemiplegia is more permanent as a rule Discovery of a potential source of an embolus makes the diagnosis more certain In a patient giving a typical history of cardiac ischaemia the occurrence of cerebral embolism is good evidence of cardiac infarction involving the left ventricle. Occasionally a source of the embolism cannot be discovered in the heart or aorta, and it may have arisen in the venous system In this circumstance the embolus has crossed from the right to the left side of the heart through a congenital opening in the auricular or ventricular septum, and the terms crossed or paradoxical embolism have been applied to the condition. Whenever cardio-arterial disease is excluded in a patient with a systemic embolism it probably belongs to the paradoxical type, if venous thrombosis in the leg and pulmonary infarction are antecedent conditions, the presence of paradoxical embolism may be assumed

Pulmonary embolism—The symptoms associated with pulmonary embolism are catastrophic in regard to their sudden appearance and their severity. They may resemble closely the symptoms of cardiac infarction ; severe dyspnoea, tightness in the chest, apprehension, small pulse, profuse perspiration, ashen pallor and low blood pressure, are the common symptoms, but the younger age of the patient,

the more severe dyspnoea, and the finding of evidence of venous thrombosis, will lead to a diagnosis of pulmonary embolism. Triple heart rhythm from addition of the third heart sound is a useful physical sign in support of the diagnosis.

If the patient survives the embolism, pyrexia is usual, haemoptysis may take place, and localizing signs in the lungs appear with characteristic radiological changes Irregularities are often found in the limb lead electrocardiogram, sometimes right bundle branch block is present, and occasionally the changes (T_2 and T_3 inversion) simulate those identified with posterior cardiac infarction. Chest lead tracings, however, help to differentiate between the two conditions.

Retinal embolism.—Sudden blindness in one eye is often the first indication that a patient has mitral stenosis. It often happens at rest so that the influence of exertion cannot be regarded as operating if this is debated during litigation. Retinoscopy shows a characteristic pattern, a cherry-red spot is seen at the macula and the adjacent retina is pale from the ischaemic effects.

Coronary embolism—Cardiac infarction from coronary embolism produces the same clinical effects as those following thrombosis It is, however, much less common, and since bacterial endocarditis is the usual source of the embolus, its interest is more pathological than clinical.

Abdominal embolism.—Splenic embolism may give rise to left-sided pain and sometimes to splenic enlargement, but in itself the condition is of no great consequence. Mesenteric embolism is a major abdominal catastrophe calling for laparotomy and resection of the infarcted gut. The outcome of this complication is naturally precarious, although recovery sometimes takes place in the absence of surgical interference. Renal embolism may be ushered in with pain in the loin and haematuria, or it may be silent Hypertension has been observed to follow renal embolism involving one or both kidneys, and the mechanism seems comparable with experimental hypertension produced by renal ischaemia.

Peripheral embolism.—An embolus lodged in one of the arteries to an extremity is a large one and consists of a thrombus. As a rule it passes the openings of the innominate, left common carotid, and left subclavian arteries, and may settle at the bifurcation of the aorta, or become impacted in one of the iliac vessels, but more often it reaches the termination of the common femoral artery. Only about one-tenth of these emboli pass into the upper extremities At the moment of impaction there is severe pain. As the collateral circulation develops, the pain tends to move towards the periphery as does the upper limit of ischaemia, and these facts are to be considered when locating the site of the embolus. Pain, pallor, and paralysis with absent pulsation of the vessels below the site of impaction are the classical clinical features of this emergency. The point where pulsation ceases, decided by palpation and oscillometry, is the most reliable physical sign of the site of impaction, and this may combine usefully with the recognition that emboli tend to lodge at sites of bifurcation or at the commencement of large branches.

TREATMENT

Since the material forming an embolus is so often a thrombus, it follows that the prevention of embolism is closely related to the prevention of thrombosis, and it

of cardiac enlargement, and embolism is a more common event in early than in late cardiac infarction in which cardiac aneurysm has developed. Pulmonary and systemic embolism is not uncommon in prolonged hypertension, but less common than in mitral stenosis It is unlikely that embolism in mitral stenosis, in which it is pulmonary as often as systemic, occurs only when certain criteria are present, and there are many exceptions to any fixed views which might be held on the relationship between the two conditions Nevertheless, clinical experience and the findings at necropsy permit certain general statements on the circumstances which appear to influence the formation of thrombosis and embolism in mitral stenosis. In certain instances of ventricular thrombosis there is evidence that embolism follows soon after the thrombosis, but no such information is available for auricular thrombosis The effect of auricular fibrillation on the formation and dispersal of a thrombus is also uncertain. The higher incidence of embolism in patients with fibrillation may be explained by the longer duration of the valvular lesion in those presenting an abnormal rhythm, and not by the actual arrhythmia Furthermore, embolism is common in cases which preserve a normal rhythm. In patients in whom *embolism develops within a short time of a return of fibrillation to sinus rhythm* during quinidine therapy, it is right to attribute the complication to the change of auricular action

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must supervise the patient's condition throughout the operation period No attempt will be made here to describe the surgical procedure in embolectomy, but it is intended to give the fullest emphasis to the need to prepare for the immediate treatment of this common and serious complication among patients in the wards of any hospital. Every hospital should nominate four members of the medical staff to form a team to carry out embolectomy in embolism The team, consisting of a surgeon and his assistant, and a skilled anaesthetist and his assistant, should rehearse all the details of the operation. The surgeon should practise the rapid approach to all the common sites of embolus impaction, in the cadaver, and be familiar with arterial surgery involving the cautious use of heparin The anaesthetist and his assistant should have a prepared plan for resuscitation in the face of circumstances prevailing during the progress of the operation Instruments required for embolectomy must be assembled and kept in readiness for immediate use. Machinery for the urgent summoning of the members of the team, for the immediate transfer of the patient to the operating theatre, and for the quick array of all equipment necessary for the emergency, should be set up and tested frequently. Other forms of arterial surgery can be allocated to the same team. Only when these suggestions are taken up in earnest will the high mortality rate now identified with peripheral embolism, and especially with pulmonary embolism, be reduced appreciably.

CHAPTER 17

THE LIFE AND LIVELIHOOD OF PATIENTS WITH HEART DISEASE

CONCERN about the home conditions, employment, and financial station of a patient with cardiovascular disease, is not part of the contemporary urge for planning in medicine, nor is it born of a new ideology. For long enough it has been noticed how quickly a patient, benefited by his stay in hospital, deteriorated after returning home, or how the worry of unemployment and insecure livelihood has hindered the progress of a patient under medical treatment either in hospital, or at home during the convalescing period The roots of the evil have been plain to see, but their removal has been difficult. In order to deal with the difficulties arising in individual patients attending a cardiac clinic, the help of an almoner has proved invaluable, but until the assistance arranged for such patients has a practical national sympathy, the benefit can only have a limited application. The time is overdue for enlisting co-operation between the State, the captains of industry, and the doctor, in order to provide for a patient with heart disease, employment commensurate with his physical ability, financial aid sufficient to relieve him of undue anxiety, and medical supervision according to his need.

It is proposed here to discuss the subject in relation to the child patient, the adult male patient, the adult female patient, and the elderly patient.

The child patient

Much trouble in the management of the adult patient could be avoided if proper attention and advice were given at the start of life. In the first place, we need to be reminded of the common incidence of rheumatic fever and chorea during school life, of our ignorance regarding its aetiology, and of the unfavourable outlook once it has affected the heart. The problem of its prevention is bound up with that of its causation, and before progress is made in this direction compulsory notification of the infection should take place, indeed, this measure has been too long postponed. If all cases were investigated and treated in accordance with a proposed plan, it is likely that the sequelae of the infection would be less frequent or less severe

When a long stay in hospital has been prescribed for a child with rheumatic fever, with the object of either preventing heart damage or limiting its effects, it is necessary to emphasize the importance of educational as well as medicinal treatment Every hospital designed for the care of children convalescing from the effects of rheumatic fever should be a school as well, and each of its wards a class-room Instruction and training must naturally be conjoined with judicious rest under medical supervision. Too often in the attempt to transfer an adult with rheumatic heart disease from heavy manual work into a clerical post, it has been discovered that the patient could neither read nor write. The refitting

of such patients into industry depends so much on their ability to succeed in a sedentary occupation that the need to emphasize the function of the school-hospital is obvious.

In the case of a child with heart disease attending school, educational instruction should be directed towards equipping him for a vocation involving the least physical exercise and commensurate with his ability to perform such work without breathlessness or undue fatigue. The propensity of the child has to be considered, and if this precludes entry into a clerical post he should be trained for a trade which may be followed without exertion and while seated. The willing acceptance by all standard industries of the services of such patients should become custom. When this gesture becomes recognized by all employers, the choosing of a career for the child-patient will have been greatly facilitated

Another problem looms large in the case of schoolchildren, it concerns the equivocal signs, particularly auscultatory signs, discovered during regularized school medical inspection. When murmurs of doubtful interpretation are met with, and they are common in young subjects, indecision leads to cautious action which lays the basis for cardiac invalidism, unwarranted as it must be in the case of innocent signs. It is not difficult to avoid this contingency. The names of all schoolchildren presenting doubtful signs should be noted by the central office receiving the medical reports, and the cases detailed to attend in convenient numbers at a heart clinic for examination by a cardiologist who has the help of devices necessary for the examination. Experience at one such clinic has shown that two-thirds of those attending have been reassured of the absence of organic heart disease Such examination aims at ensuring proper direction at the start of life and preventing the pitiable consequences of unwarranted cardiac invalidism.

The adult male patient

The problems presented by the adult male patient with heart disease are many, and they include the transfer of a patient from a heavy to a lighter industry, training for such newer occupation, compensation for lessened stipend, the upkeep of a family, allowances, and medicinal treatment sometimes involving travel over long distances.

The diversity of the problems makes it impossible to prescribe general rules to govern the environment of all patients with heart disease, and serves to illustrate how indispensable are the services of a trained almoner at any centre supervising the treatment of such patients A short description follows of the troubles met with in the case of a man aged 52, with mitral stenosis and auricular fibrillation, during a stay in hospital for treatment of an exacerbation of heart failure, and of the steps taken to remedy those troubles

He had followed his occupation as a crane-driver for 23 years, and for 7 years he had attended a hospital's out-patient department on account of fibrillation and heart failure Latterly mercurial diuretics had to be added to digitalisation and twice he had received in-patient treatment During this third admission he was questioned about any worries he might have, and the almoner was asked to visit his bedside That week he had been summoned for default of payment of "separation money" to his wife; legal aid was enlisted on his behalf and the summons was withdrawn The county authorities also were suing him for default of payment in respect of his children evacuated to the country because of enemy air-raids, when they were told of his illness they withdrew the claim As the time for his discharge drew near a civic welfare committee was informed, and since both his wife and children were away the committee arranged for the cleaning and heating of his house for his

home-coming and during his convalescence He had been advised on previous occasions to forego his heavy work, but he had persevered because of his financial responsibilities in the home, application to place him in a lighter job was made to his employers, but this proved unsuccessful, in view of his faithful service during 23 years his employers were then asked whether they would award him a pension, but they only subscribed ten shillings a week In his shed in the garden the patient had a lathe, so the almoner approached the Ministry of Labour and, since the patient was able to work to specification detailed on a blue print, occupation was provided for him, this brought him £4 per week, working shorter hours, involving little exertion and no travelling Lastly, the almoner found the shed unheated so that an electric fire was installed before the winter set in Such help restored happiness to the patient, and enabled him to meet his financial responsibilities, while continuing with medication by means of digitalis and mercurial diuretics.

The adult female patient

The responsibility of the home, involving housekeeping, laundering, shopping, and preparations of meals, affords little rest for the female patient with heart disease. Much can be done to ease the burden, and foremost is a proper understanding by other members of the family of the nature of the illness and the need to take adequate rest Pregnancy supplies another problem peculiar to this group, the birth should take place in hospital; other aspects of this subject have been dealt with elsewhere in this book The almoner can also give useful information on certain procedures which will provide opportunity for resting; she can advise the patient on how to obtain home-help, suggest school meals for the children, arrange for hot meals to be sent to the patient from invalid kitchens, and can place the patient in touch with the various voluntary societies who are willing to help in diverse ways.

The elderly patient

In this group, too, the almoner's advice can ease the responsibility of the cardiologist in treatment. This will be directed towards finding a home-help, arranging for allowances from other members of the family, giving information about pensions to which patients may have a claim, and organizing medicinal treatment either at home or at hospital.

What has just been written is not to be regarded as a whim in sociology. It describes accessory means in the treatment of heart disease and heart failure, which, if withheld or withdrawn from the familiar therapeutic agents like digitalis and mercurial diuretics, will rob the results of regularized medicinal treatment of a great measure of success.

HEART DISEASE IN RELATION TO INDUSTRY

Cardiology has always included an opinion on a patient's occupation which would prove most favourable to the heart under the handicap of a particular disease. In this department of medicine at least, it has been customary to protect *the patient against undue exertion on the one hand and, on the other, to help him* to gain work which would ensure his livelihood. The present need is to establish a closer liaison between doctor and almoner, and in order to cement this co-operation, a scheme should be devised which would supply a classification of industrial health Such a classification should be made available automatically to the almoner, who should, in turn, interview the patient and advise and plan for his benefit. It has been found satisfactory to arrange patients in four groups in accordance with their ability to undertake physical exercise or exertion;

TABLE XV

Patients arranged in four groups according to the amount of physical exercise non-injurious to their particular cardiac disability

Industrial health category	*Nature of heart affection*	*Suitable occupation*
NW	Intractable heart failure Controlled heart failure, coupled with infirmity of old age	No work
SW	Mitral stenosis Aortic incompetence Controlled heart failure	Bank clerk ; accountant , short-hand typist , other clerkships Librarian Draughtsman , architect , designer ; commercial artist Proof-reader for newspaper or publisher Tailor ; dressmaker , embroiderer Electrical bench worker, fountain-pen maker and repairer , toy and cigarette lighter repairer Despatch clerk and packer of light goods, for example, drugs Inspector of factory goods on conveyor-belt system Telephonist
LW	Aortic stenosis Cardiac ischaemia Cardiac infarction Hypertension Controlled heart failure	Post Office sorter Factory store-keeper Radio mechanic Printer's compositor Van driver without loading or unloading Shop assistant Tailor's fitter Surgical and precision instrument maker Jig and tool maker Glass blower
AW	Unwarranted cardiac invalidism from erroneous diagnosis	Any work

it is felt that the designation applied to each group should not convey to the patient any hint as to grading lest an erroneous interpretation should be given to it. This is avoided by nominating the groups as NW (no work), SW (capable of sedentary work with travelling to and from work), LW (fit for light work involving some exercise but avoiding exertion, and AW) (capable of undertaking any work) (Table XV).

Decision as to the class to which a patient is allocated comes from a consideration of the natural history of the illness and the tolerance of a particular cardiac disability to the physical exertion demanded by a particular employment. The need to apply such a classification is greatest in children of school age so that they might be directed towards, and trained for, the suitable calling nominated for them Of equal importance is the care necessary to avoid unwarranted invalidism imposed by an erroneous diagnosis. Indeed, it is likely that there are as many false invalids capable of undertaking heavy industry as there are true invalids needing withdrawal to lighter occupations. Our watch for the former category should be as intent as for the second, knowing the injustice which results if diagnosis miscarries Under the heading of *Unwarranted Cardiac Invalidism* it is necessary to recount with emphasis those conditions which commonly lead to such invalidism.

UNWARRANTED CARDIAC INVALIDISM

"Angina"

Pain over the heart is seldom a heart pain. Discomfort at the left breast is so often a sign of an overwrought nervous system that allusion to a precordial site should be rejected on the grounds that a pain here does not bear any relation to the heart. A sternal distribution for the pain makes it likely that it has its birth in the coronary circulation, either from cardiac ischaemia or from cardiac infarction. Apart from the site of the pain and its ready onset after exertion, a guide as to its origin will come from the way in which the patient describes his symptoms both in mannerism and in aphorism. The patient with coronary disease has no desire to emphasize, and far less to exaggerate, his symptoms. He tells of his pain, which is never of the lancing kind, quietly and without recourse to rhetoric. When such things are kept in mind, and help from the electrocardiogram is sought more often, the false label of "angina," with the hardship and handicap which it imposes and the seriousness that it implies, can be withdrawn from numerous erstwhile invalids.

"High blood pressure"

It is true to say that this designation is being misapplied to countless healthy subjects, and when medical examination becomes the condition of general employment the number is likely to swell. This state of things has resulted from want of a clear definition of hypertension, and from attention to the blood pressure reading by itself, usually obtained during the natural excitement and anxiety of a first medical examination, and without notice of the presence or absence of cardiovascular hypertrophy found by clinical and cardiographic examination, providing pathological evidence of hypertension. It is likely that a raised blood pressure has to exist for a time before cardiovascular hypertrophy shows itself, but there is not any proof as yet that the higher and labile values are

prodromal signs of hypertension, and the warning is needed to avoid applying a diagnosis of hypertension when a moderate rise of the blood pressure above the orthodox value is the only physical sign.

"Heart enlargement"

An apex beat found outside its customary position has been too readily accepted in the past as evidence of cardiac enlargement. The commonest explanation of the displaced apex beat is the shifting of the healthy heart from any cause, notably scoliosis, and it should become a custom, when other signs are equivocal, to look for this mild deformity by inspecting specially the set of the shoulders This simple habit in clinical examination will by itself save countless healthy subjects from unwarranted invalidism.

Even when moderate enlargement of the heart shadow has been noticed on radiological examination, the presence of simple bradycardia or of depression of the sternum must receive first attention as possible benign causes, before judging the effects to be the outcome of disease. It can be said that an examination which at its close offers " enlarged heart " as the only heading in the diagnosis is incomplete and imperfect, and for that reason it is mischievous.

"Tired heart": "Strained heart"

A heart free from disease cannot tire and it continues to beat at a force and frequency customary for it. Similarly, under the stress of sudden or sustained exertion the heart remains unalterable except that it naturally increases its rate in keeping with increased metabolism in the rest of the body. To explain the symptoms of a patient on the grounds of a " tired or strained heart " is not only improper, but may cause great harm.

"Fatty degeneration": "Fatty heart"

Language necessary to the pathologist at the microscope to describe changes which by themselves can hardly interfere with cardiac function cannot serve for the clinician at the bed-side, and he must forego the terminology, for it will mislead in diagnosis. In a fat patient to think of a fatty heart is fantasy.

"V.D.H." and "D.A.H."

The use of the terms " valvular disease of the heart " and " disorderly action of the heart " is becoming unfashionable, and they are mentioned here in order to expedite their exodus. Both terms are unacceptable because of their vagueness, so that if V.D.H. is present, the valve and the nature of the injury have to be named, while *in the case of D.A.H. the rhythm has to be nominated as extrasystoles,* auricular fibrillation, and so on. If such ambiguity in diagnosis has to exist in regard to a patient, it should not be for long, and the help of another practitioner must be sought who will decide in the light of clinical experience or with the aid of special tests

Unwarranted invalidism is also commonly born from the misinterpretation of healthy splitting of the first heart sound for the presystolic murmur of mitral stenosis, or of the innocent systolic murmur for mitral disease. The latter mistake often arises from the irresponsible use of the term " mitral incompetence "

to explain any murmur in the mitral area, and it adds fervour to the plea that the term should be excluded from clinical diagnosis on the grounds that it hinders an accurate exposition of it.

"Heart failure"

Oedema of the ankles has been too hastily accepted in the past as evidence of heart failure When the mistake has been made in the absence of heart or renal disease it has usually meant that innocent signs have been misinterpreted, or that oedema has been an incidental finding in a patient breathless from emphysema. In those with heart disease the onset of oedema from causes other than heart failure needs more care than usual, and radiological examination of the pulmonary vessels may be necessary before a decision is made.

"Effort syndrome"

This has been dealt with fully in the context, but it bears repetition here that should a subject carry the bizarre symptoms described for this condition and be excused from any kind of service on that account, it should be known that such exemption is on the grounds of neurosis, the stigma of which may become so repellant to the subject as to help him to overcome the symptoms. The heart should not receive any mention in connexion with "effort syndrome" unless for thepurpose of making it clear at the outset of a discussion on the condition that it does not bear any relation to the heart.

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